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ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY
ARISING FROM THE USE OF ASBESTOS IN ONTARIO

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180 Dundas Street
Toronto, Ontario
Friday,
June 19, 1981

VOLUME XI



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ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY
ARISING FROM THE USE OF ASBESTOS IN ONTARIO

VOLUME XI

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180 Dundas Street
Toronto, Ontario
Friday,
June 19, 1981

VOLUME XI

180 Dundas Street
Toronto, Ontario
Thursday,
June 18, 1981
Volume XI

THE FURTHER PROCEEDINGS OF THIS INQUIRY
RESUMED PURSUANT TO ADJOURNMENT

APPEARANCES AS HERETOFORE NOTED

DR. DUPRE: Have the parties selected an order?

MR. HARDY: I believe I'm starting, Mr. Chairman.

DR. DUPRE: Mr. Hardy, if you please, sir.

DR. GEOFFREY BERRY, PREVIOUSLY SWORN, RESUMES THE STAND
CROSS-EXAMINATION BY MR. HARDY

MR. HARDY: Good morning, Mr. Berry.

MR. BERRY: Good morning.

MR. HARDY: Q. Yesterday there was a little bit of discussion of article number nine, Pathology and Mineral Content of the Lungs in Cases of Mesothelioma in the United Kingdom in 1976. I gather in that study in which you worked, Mr. Berry, the lungs of both mesothelioma cases and persons apparently without asbestos-related disease were measured for fiber content?

THE WITNESS: A. Yes.

Q. On table...figure two on page 193, there is an indication that in the two control groups with no apparent occupational exposure, that you knew of, to asbestos, asbestos of various fiber types was found in the lungs of a good percentage of the persons?

A. Yes, that is correct.

Q. Is it your understanding from where the patients came that these exposures were likely to be from general background?

5 A. Well, the controls were chosen by...they were supplied by the pathologist who supplied the case, so that they came from an area where a mesothelioma had been known to occur, and therefore they would be selectively drawn from those areas where asbestos is used - because mesotheliomas had occurred. So in that sense they would be more likely to have been exposed to asbestos than a completely random sample of the general population of the United Kingdom.

10 Now, of course, I did say...I think I mentioned yesterday, we have done a study in 1977 as well, where we haven't chosen control material in that way. We have chosen it by taking consecutive post mortems from a number of towns in the United Kingdom, including some which were towns in nonindustrial areas, and we obtained a fairly similar result.

15 Q. Meaning that you found asbestos fibers of various types in the lungs of, among other persons, rural residents where there is not necessarily industrial asbestos used?

20 A. Yes.

Q. Has amosite been used extensively in the United Kingdom?

A. It has certainly been used quite a lot, yes.

25 Q. Because I noticed that some of the persons in the study we are looking at here had amosite found in their lungs, some of the control group persons?

A. Yes.

Q. As well as chrysotile and crocidolite?

A. That's correct, yes.

30 Q. Asbestos is a naturally-occurring mineral found in many parts of the world?

A. Yes.

Q. And I would guess that it has been in the general human environment for many, many years. Is that true?

A. I would think so, yes.

Q. I would guess in part that's due to erosion of rocks, wind, water carrying natural asbestos?

A. Yes. Well, you know, I don't know much about how much asbestos is generated by those processes you described.

Q. But we do know that asbestos has been found in the lungs of a large, of a varied group of persons throughout England, not necessarily related to living near or working in asbestos operations?

A. Yes. Yes, but I'm not aware that we have any situations where asbestos would be liberated from rocks by erosion. I mean, maybe we have. I just don't know. We certainly haven't got any areas where we mine asbestos.

Q. In the United Kingdom?

A. That's right.

Q. Could we turn back a page to page 192 of this article number nine, and look a little at table five at the bottom of that page? I believe it indicates that among both cases and control studies a number of additional asbestos fiber types were found in the lungs, namely anthophyllite, tremolite, actinolite? Is that correct?

A. Yes. Yes, they were found in a minority of people.

Q. A minority, but maybe a substantial minority. It appears that somewhere between twenty and forty percent various differences, differences for different fibers in different groups, but somewhere in that range?

A. Yes. Right.

Q. Asbestos fibers were found in the lungs?

A. Yes.

Q. Is it your understanding that those three types of asbestos fibers are generally not used commercially?

5 A. Well, anthrophyllite has been used commercially. It used to be mined in Finland, but that mine is now closed down. Tremolite and actinolite, I don't think are used commercially at all, although tremolite does occur as a contaminant of chrysotile in Canada, and I think also of talc in the United States.

10 Q. Are you aware, Mr. Berry, of a number of substances, naturally-occurring substances found in the general population, which have been found to be carcinogenic at high doses...other than asbestos?

A. Yes, I'm aware of that problem in general. I can't just think of the materials offhand, but, yes.

15 Q. Selenium might be one example?

A. Sorry?

15 Q. Selenium? Is that a naturally-occurring element?

A. I don't know anything about selenium.

20 Q. I guess lead has been...do you know that lead which is found naturally in the human body has also been found to be carcinogenic at high doses, in various sorts of studies, in animal bioassays?

A. I don't know offhand, no.

Q. Excuse me?

25 A. No, I didn't know that lead had been shown to be carcinogenic in animals.

25 Q. But you are aware of the general finding that some naturally-occurring substances at high doses have been found to have adverse health effects...

A. Yes.

Q. ...including carcinogenecity?

30 A. Yes.

Q. You were talking yesterday to some extent

5 Q. (cont'd.) about acceptable risk and determining safe levels. Isn't it relevant in that sort of determination to consider the fact that some substances which are very useful at high doses, are very dangerous at high doses? Can that totally be eliminated by the human population, because they are naturally occurring in the environment?

A. Yes.

10 Q. So we can never be totally safe if we believe that even the minutest amount of that substance may pose some danger?

A. Yeah, I would agree that we can never be totally safe, yes.

15 Q. There was a good deal of discussion yesterday of dose-response relationships, and crucial to the dose-response relationships is the calculations which have been made of dose in the studies from which the relationships are generated?

A. Yes.

20 Q. I would like to look a little more closely at some of the dose information in some of the studies you have worked on, and maybe if we could turn first to your 1979 article in the British Journal of Industrial Medicine, on Asbestosis at the Rochdale Plant, which is number eight.

If we could, please, look at the table number one on page 101? I believe table number one indicates the dust exposures used to determine the dose-response relationships in this paper?

25 A. Yes.

Q. Which show, among other things, that the mean dust level in 1936, in this plant, was believed to be thirteen point three fibers per cubic centimeter?

A. Yes.

30 Q. Even as recently as 1951, it was believed that the mean dust level was ten point eight fibers per cubic centimeter?

A. Yes.

5 Q. Do you have any understanding of what mean dust levels are likely to be in plants in the United Kingdom or the United States today, or Canada today, which are complying with the two fiber per cubic centimeter standard?

A. Well, I haven't got a great deal of idea, no. But if the plant is complying with the two fiber standard, then the mean is probably around about one.

10 Q. Which would also mean that a worker in 1951 in this Rochdale Plant, based on the exposure data used here, would have had as much exposure in one year as a current worker would get in ten years?

15 A. Yes, that is so...possibly more, because we were talking yesterday about changes in measuring slides that had been collected in dust sampling instruments, and the graticule method. I don't know if I made it clear yesterday that these figures are the pre-graticule method, so that nowadays these would probably be scaled at my factor of two.

20 Q. I was planning to get to that, and why don't we do that right now? Are you aware of an attempt by Julian Peto, who has also done work with the Rochdale factory, to take these data on exposure and convert them to what they would have counted with the new graticule counting method?

25 A. Yes. He gave that at the Lyon Conference, I think. Yes. His earlier papers used these data, but then later he was working, I believe, with the hygienists in the factory at the time when they were investigating the changes due to graticule counting, and he altered the figures accordingly... according to what was found.

30 MR. HARDY: Mr. Chairman, I have a copy...as a matter of fact, I have several copies, of the paper where Mr. Peto did that alteration, which has a table in it which I think might be very useful for us to use.

DR. DUPRE: Do you wish to enter that as a numbered exhibit?

5 MR. HARDY: I would be glad to. It's also among the documents which Julian Peto has submitted to the Commission.

DR. DUPRE: How do you want us to deal with it for the record, counsel?

10 MR. LASKIN: Let's put it in now. It will in fact be part of Mr. Peto's brief that the Commission will prepare, but if Mr. Hardy is going to refer to this, I think we should put it in now.

MR. HARDY: I've got some copies here. Maybe we could give a copy to the witness, maybe to the chairman.

Would you like to mark this as an exhibit number, counsel?

15 MR. LASKIN: Fourteen.

EXHIBIT # 14: The abovementioned document was then produced and marked.

20 MR. HARDY: Q. Perhaps if we could turn to page 832 of exhibit fourteen.

Now, to make sure I understand, Mr. Berry, what it is that Mr. Peto did in this document. As I understand it, current counting methods for dust monitoring count more fibers on a fiber (sic) than would have been counted on that same exact fiber by the techniques used twenty years ago?

25 THE WITNESS: A. Yes.

Q. I guess as you put it yesterday, there has in effect been a tightening of the two fiber standard, totally because of technical changes in the way monitoring is done?

A. Yes.

30 Q. As I understand it, what has been done here in table two is that the previous estimates of exposure for the

5 Q. (cont'd.) Rochdale plant, which are in the top line, have been studied and attempts have been made to revise those estimates to reflect how the exposure levels in the Rochdale plant in previous years would have been measured if counted with today's methods?

A. Yes.

10 Q. If we look at the top line of table two, I believe those are the same numbers that we saw in your paper, is that correct?

A. Yes.

Q. For instance, in 1951, the assumption was that the mean dust level in the plant was ten point eight fibers cubic centimeter?

A. Correct.

15 Q. The recalculation in the Peto paper is that by today's counting methods those exposure levels would have averaged thirty-two point four fibers?

A. Yes.

Q. Which I guess is a factor of just about exactly three?

20 A. Yes.

Q. Similarly, the 1956 figure has gone up from five point three fibers per cubic centimeter to twenty-three point nine fibers per cubic centimeter, which is an increase of about four times?

A. Yes.

25 Q. Have you been involved in any of this recalculation effort, Mr. Berry?

A. No.

30 Q. But talking as we were a minute ago about the man in 1950, compared to the man in today's plant, based on these newly recalculated figures a man exposed for one year in 1951 in the Rochdale plant would have more than thirty times as much

Q. (cont'd.) exposure as the man exposed for one year in a plant today complying with the two fiber standard?

A. Yes.

Q. Yesterday you talked about...you had mentioned this change in monitoring techniques, and you also mentioned another difference in monitoring techniques between the past and the present. Do you recall what ...the other difference?

A. Well, it was personal sampling, was it?

Q. Right. Could you explain the difference between static sampling and personal sampling?

A. Well, a static sampler is a sampler situated near where people are working or near where machines are operating. As the word implies, it stays there throughout so it measures the concentration of dust in that particular area. So, for example, if we were to imagine we were exposed in this room, we might put one on here. One would then estimate, in applying this to individuals one would say that the people who...all of us sitting around here were exposed to the dust measured there, and you might have another one in that corner and say that because you are halfway between, for you we'll take the average, and for the chairman and myself and the commissioners, we'll take the value here.

Now, for a...and of course in a working situation it's more complicated than that because everybody is not sitting still, they are walking about.

A personal sampler is something that a man wears on his chest, and therefore as he moves about it samples the air wherever he happens to go to. So it gives a more accurate measure of the exposure of that particular individual.

Q. As I understand it from your discussion yesterday, there has been some findings in the United Kingdom which I believe were referred to, among other places, in the Simpson report, to the fact that personal samplers typically

Q. (cont'd.) monitor higher levels of dust than stationary samplers. Is that your understanding?

A. I seem to recall that, yes. I can't recall by how much.

Q. That was going to be my next question.

Though you don't recall how much, it is a fact that personal samplers monitor higher dust levels and the current plant we are testing for compliance with the two fiber standard, through use of personal monitoring, the plant would have to be even less dusty to comply with the two fiber standard than if they were doing their compliance monitoring with just static samplers?

A. Yes. Yes, I think that is correct.

Q. These Rochdale exposure figures, both the numbers you used and the numbers that have been recalculated in the Peto document, I gather are based on static sampling?

A. Yes.

Q. Where a further recalculation attempted, based on what those levels of exposure were at the Rochdale plant in these previous years based on personal monitoring, the numbers would go up even more, I gather?

A. The dust levels?

Q. That's right.

A. Yes.

Q. So we would be talking about an even greater discrepancy between the dust levels historically at Rochdale and the dust levels in a plant today which was complying with the two fiber standard and doing it by use of personal monitoring?

A. Yes.

Q. In the other of your cohorts...both of the other cohort studies that you have been involved in, there is also some...although less-detailed discussion, of the dust levels?

Maybe we could look at what Widdle has stated

5 Q. (cont'd.) about the East London factory dust levels...and I think if we look at paper number six, which is your New York Academy presentation in 1979, entitled Patterns of Mortality in Asbestos Factory Workers in London...there is some information on exposure levels in that plant. Do you recall the discussion in that paper?

A. Yes. This is on the first page, isn't it, of that paper?

10 Q. Right. Perhaps you could tell us what you were able to say then, and if you know anything more now what you can add about the likely dust levels in that East London factory?

15 A. No, we certainly don't know any more now, and in fact we...this information only came to light relatively recently, just before this meeting in fact, this meeting in New York, and previously to that we had no information on fiber levels at all. These figures were produced by...I've forgotten the name of the gentleman, but somebody who had been manager of that plant and knew what was going on and what measurements have been made, and these are the figures that he produced. 20 But they weren't based on very many measurements, from what I recall, and we don't know quite how the sampling was done.

25 Q. But I gather the information, sparse as it may have been, led you to suggest that prior to 1945, those exposures which you had labelled severe in the work on this cohort averaged twenty fibers per cubic centimeter or higher?

A. Yes.

Q. Then, I guess, that further information was that for those jobs you would consider low/moderate, the average was somewhere between five and ten fibers per millilitre?

30 A. Well, yes, although there was the...there were also the nonproduction jobs which would end up in the low/moderate..well, they would be in the low category, which

A. (cont'd.) would combine with the moderate, and there they were less than five fibers.

5 Q. As I read those numbers, the severe exposures were in this Cape Asbestos East London factory, were higher than the exposures that we were talking about at Rochdale a minute ago?

A. Yes. Yes, they were at least fifty percent higher, based on these figures...possibly double.

10 Q. I assume that the figures in the 1979 paper on the East London factory were based, like your Rochdale table, on the old counting method?

A. Yes, they certainly would be, because they related to samples that were being counted many years previously.

15 Q. Were we to correct those exposure numbers to the current fiber counting method, as has been done for the Rochdale data, they would be two to four times higher exposure numbers?

20 A. Probably. We can't say for certain, because in contrast to the Rochdale data we were looking at a minute ago, those calculations were done entirely within the Rochdale factory, so we know the factor is three, about threefold for the method used at Rochdale. We know far less about what was going on at this London factory.

Q. You also have exposure data in your recent study of the friction materials plant, which is number fifteen?

A. Yes

25 Q. I believe there is a table on the second page of that article, that presentation, which provides some mean concentration figures during periods over the years at that plant. Is it true that these figures also, like the East London factory figures, have not been corrected to take into account modern counting techniques?

30 A. Yes. I can't see that mentioned. A lot of

A. (cont'd.) these measurements were done quite recently. I would expect that they were done by the modern methods, but...

5 Q. Particularly...that would be particularly the measurements between 1970 and 1979, I would guess?

10 A. Yes. Well the measurements, all the measurements were made recently, even those that refer to early periods, because measurements were only introduced into this factory in 1967, and a lot of these figures have been produced by simulating the earlier conditions, which was done by my coauthor, Mr. Skidmore, in conjunction with people at the factory, by running machines over weekends with the ventilation turned off, and things of that nature.

15 Q. So there may not be the same need for correction to modern measurement techniques with this data as there is for the other two cohorts?

A. That should be the case, yes.

Q. Do you know whether these are static samples or personal samples?

20 A. I think these are personal samples, because it's stated at the bottom of page ten point six point one that personal samplers were introduced in 1968, so I think so, but I'm not completely certain.

25 Q. If we could return to your 1979 Rochdale paper, I think I would like to talk for a couple of minutes about the importance of the changes in measurement of exposure to some of the calculations you did in that paper.

A. Yes.

Q. Perhaps we could turn to the paragraph you discussed with Mr. Laskin yesterday, on page 105 in the section entitled, Relationship of to Cumulative Dust Exposure.

30 A. Yes.

5 Q. I believe yesterday, with Mr. Laskin, you talked about the difference between the one hundred and twelve fiber years per cubic centimeter calculation, which in 1968 was believed by the BOHS study to equate with one percent prevalence of crepitations, and your recalculation in this study that a prevalence of one percent might well occur at forty-three fiber years exposure?

A. Yes.

10 Q. I believe you went through three reasons with Mr. Laskin on why that number has decreased?

A. Yes.

15 Q. If you were to further refine that estimate, based on the new exposure numbers calculated in the Peto document, historically at Rochdale, what would happen to that number?

15 A. It would increase by between two and threefold.

Q. In other words, the prediction of at what exposure level one percent crepitations would occur would go up to somewhere between eight and a hundred and thirty fiber years?

A. Yes. Correct.

20 Q. At which point the estimate would be sort of back where it was in the 1968 document?

A. Yes.

25 Q. If there is a further difference between static sampling and personal sampling, that number would go up even higher in terms of a current plant complying with the two fiber standard, using personal samplers?

A. Yes, that is so.

30 Q. In addition in this paper, you discussed the alternative models for expressing exposure over the years, talking about both the cumulative exposure model, which is the one that has traditionally been used, and an exposure model which weights time as part of the cumulative exposure?

A. Yes.

5 Q. I wanted to talk about that in a slightly different way than it was discussed yesterday, and let me ask it this way: If we have two men, both of whom started work in an asbestos plant in 1950, and the first man worked in that plant for one year and there was an exposure level of thirty fibers per cubic centimeter, and the second man worked in a different factory, say, for thirty years from 1950 to 1980, at an exposure level each year of one fiber per cubic centimeter, based on the traditional cumulative exposure model both men would be assumed to have had the same dose of asbestos?

A. Yes.

15 Q. But under your alternative theory, or the theory of Jahr, the man exposed for one year at thirty fibers would be considered to have a much more significant dose of asbestos?

A. Yes, that is correct.

20 Q. Which would mean that the first man would be expected to be at much greater risk of developing disease, I assume?

25 A. Yes, and we could calculate it out on the Jahr model. The first man would have thirty fibers thirty years ago...that's thirty by thirty, that's nine hundred...and the other would have one thirty years ago, one twenty-nine years ago and one twenty-eight years ago, etc., so it would be the sum of, first, thirty integers, which is...what is that...four hundred and sixty-five, I think.

So one would have double the dose of the other, in Jahr's terms.

30 Q. So that if that alternative model is correct, there is an additional factor to consider in comparing the Rochdale cohort experience to the man currently employed in a factory that is complying with the two fiber standard, which

Q. (cont'd.) is that those high exposed many years ago may have undue, as we just exemplified, twice as much, importance in determining disease?

5 A. Yes, that's right. That's the basis we use for making these alternative calculations which we were discussing yesterday.

Q. Let me just return, just a second, to another thing in this dose-response area, which is that you showed a slide yesterday where you compare standard mortality ratios for lung cancer, assuming that fifty fiber year exposure, based on a number of studies...

A. Roughly a hundred fiber years.

Q. Right. Okay. One of the numbers was for the Peto study, which is the mortality study at the Rochdale plant?

15 A. Yes.

Q. I gather that the number that you showed was uncorrected for the next exposure calculations that Julian Peto has done?

20 A. No. I think that was corrected. I think that was from the Lyon paper that you have given as an exhibit.

Q. I think the number on the slide, as I recall, was a hundred and sixty - was the SMR?

25 A. That's correct, and I got that from page 833 of exhibit fourteen, where he talks of, in the first paragraph, "a cumulative exposure of between two and three hundred", so I called that two hundred and fifty, "an average SMR for lung cancer of between two and three", I call that two point five. I then fitted a line we've got...we've got a point at two fifty and two point five, and at zero it would be one point nought, draw a line like that and at a hundred that's two-fifths of the way from there, two-fifths of one point five is nought point six, so that's one point six. It's similar

30

5 A. (cont'd.) to some figures that Peto had given himself in earlier papers, although that was before the correction and for various reasons he comes back to where he was before as well.

Q. Do you know, it's not clear to me from Peto's discussion there at the top of page eight thirty-three that he is using the corrected exposure numbers in that paragraph.

10 A. I mean the whole paper is about revising the exposures, isn't it?

Q. Well, there is certainly a section of it, but I found it a little hard to follow what he was doing, and maybe we'll just have to ask him when he comes here. That might be better than having you speculate, quite frankly.

15 A. Certainly, but I think it is pretty clear from the second part of 833, but of course you can ask...get it from the horse's mouth.

Q. Okay. We'll let him know that you gave me that opportunity.

20 Let me move to a different subject and draw your attention that some numbers that you actually put up on the blackboard yesterday, which are still up there fortunately, referring to smokers and nonsmokers in some data that Dr. Selikoff has published, and perhaps you could, as an introduction to the questions I ask, explain the numbers in the table once again.

25 A. Yes. These are from the paper that I referred in the Annals of New York Academy of Sciences, by Hammond, Selikoff and probably Seidman, I can't quite remember, and these are death rates due to lung cancer for, I think it was for a hundred thousand man years of risk. Anyway, they are all standardized as the same base.

30 We've got four combinations according to whether

A. (cont'd.) they smoked or didn't smoke, and according to whether they were exposed to asbestos or not.

5 The nonasbestos figures have come from some of Dr. Hammond's studies in the American Cancer Society, and the asbestos figures from the insulation workers.

10 Well, these are the death rates standardized so they are comparable, and if we compare asbestos workers with nonasbestos workers, looking, standardizing for smoking, so we are comparing that figure with that one, or we are comparing that one with that one, then we get a smoking effect which is about five, a smoking factor of about five, just to keep things in round numbers.

Q. Don't you mean an asbestos factor?

A. Yes. Sorry..

15 Q. You referred to five as a smoking factor. Is that what you meant to say?

A. No, I didn't. Thank you. Yes, as an asbestos factor.

20 So this particular type of work, the exposure to asbestos was such as to increase the lung cancer risk by a factor of five.

Now, smoking, we have, comparing within nonasbestos workers, an effect here of about elevenfold, and we have an effect here, six hundred and one divided by fifty-eight, which is again near enough to eleven.

25 So for the combination, if we call nonsmokers, nonasbestos, and if we just make everything relative to them, so we'll say they've got a risk of...these people have got a risk of five, these of eleven and these of fifty-five, which is five times eleven.

30 This illustrates what is called the multiplicative effect, and it was Doll in a paper in 1971, who first pointed out that Selikoff's original data, which was an earlier version

5 A. (cont'd.) of this, fitted the multiplicative model very well. It was also what Dr. Newhouse and I showed in our 1972 paper on a different set of data, and then this later followup by Hammond, et al, shows things up a lot more clearly because there's more time elapsed and more cases.

10 Q. As you were describing that information you said something which was exactly what I wanted to ask you about, which is, you said that the five factor for asbestos exposure was based...I believe you said the particular type of asbestos exposure that occurred in this cohort?

A. Yes.

Q. Could you maybe elaborate on what you meant by that?

15 A. Well, what I meant was that the five isn't a universal figure that applies to all asbestos situations. If there are...there have been cases where higher factors have occurred and obviously there have been cases where lower factors have occurred, say threefold, and so five is just the figure for their particular plant or set of insulation workers. The same as the eleven is the figure for an average smoker. People who smoke more would have a bigger factor. People who smoke just a little one much less.

20 Q. If we were looking at the number that perhaps came out of your recent study, the friction material plant, instead of five we would be talking about something much lower, I assume?

25 A. Indeed, yes. We would be talking about near enough to one, because there was no excess in that plant.

Q. But a man who is a smoker in that plant today would be facing the same eleven times risk of lung cancer as the man in Selikoff's insulation cohort many years ago?

30 A. No, no.

Q. Or as the man many years ago who is not

Q. (cont'd.) an insulation worker, but who is a smoker?

5 A. Yes. I mean, we needn't bring in many years ago. I mean he would have an increased risk of about eleven because eleven applies to British smokers as well. He would have an increased risk of around about eleven, compared with one of his workmates who didn't smoke.

10 Q. Comparing again two men in today...or taking a man today who is a smoker and who works in an asbestos plant, the way I read the numbers we are talking about getting that man to stop smoking, or hopefully preventing that man from ever smoking, would have a much greater impact on lessening his risk of lung cancer than changes in exposure levels now that we are down with much lower exposure levels than were true in the historical cohorts?

15 A. Yes. Yes, I think most people recognize that is the biggest reduction in lung cancer that could possibly occur, would be if everybody stopped smoking. There is not much sign that that's likely to happen, I'm afraid.

20 Q. But there might well be...there certainly are compelling reasons to use governmental and other resources to achieve that end?

25 A. Well, it depends what you mean by compelling reasons, but there's good evidence to show that that will be good for the nation's health, yes. I don't want to be drawn into discussions on whether governments should try and stop people smoking.

30 Q. I don't want to draw you into them either. I think the point that I was interested in is that if you look at the smoking/asbestos interaction with data from a cohort with lesser exposures than were true in the Selikoff cohort, you are going to end up with a much greater discrepancy between the eleven and the number that's attributed to asbestos exposure,

Q. (cont'd.) Isn't that right?

A. Sorry, I didn't quite follow that.

Q. Maybe I should withdraw it and move on.

5 Let me ask you then about asbestos, smoking and asbestosis. I believe in some of your papers you have found the same sort of interrelationship between smoking and asbestosis as we have just been discussing between smoking and lung cancer?

10 A. Well, we've found that smokers are at more risk of developing signs than asbestosis than nonsmokers, but when you say the same kind of relationship, we haven't considered that as a multiplicative one and indeed one couldn't because if we are talking about asbestosis then there isn't a base rate for nonasbestos workers.

15 Q. Maybe if we looked at your 1979 paper on Rochdale, which is number eight, perhaps you could describe for us some of the findings there which are in table seven, on the Association Between Smoking and Asbestosis.

20 A. Right. Well, table seven, and there is a short paragraph discussing table seven, starting on page 103 and coming over to page 107, but if we look at people first employed after 1950, and in table seven we divide these people into age groups, because the nonsmokers were younger than the smokers so we took account of age so that we weren't being misled into confusing an age effect for the smoking effect.

25 If you look at table seven, the bottom part, there are three rows which refer to never smoked and those who smoked one to four cigarettes a day, which is a pretty light rate of smoking, and there are very few signs...there were just two people with radiological signs, out of fifty-five people. None of them had crepitations or possible asbestosis, and none of them had been certified.

30 The bottom three lines refer to smokers. Now there were more of those, there were a hundred and forty, so between

5 A. (cont'd.) two and a half and three times as many, but the number with some sort of sign was far more than two and a half or three times as many. There were twenty-six, for example, with radiological changes, and there were ten being certified. Twenty-six have got crepitations, and thirteen - possible asbestosis. So the smokers seemed to be at a much greater risk.

10 Q. I gather that one of the possible ways of interpreting that data would be that if a man stops smoking that would have an impact on making it less likely that he develops asbestosis, or if a man never smoked it would make it less likely that he develops asbestosis?

A. Yes.

15 Q. Have you looked at relationships between asbestosis and smoking in any other groups of workers?

A. No. This is the only group where I've actually looked at asbestosis as opposed to mortality. All the other studies I've been involved with have been mortality studies.

20 Q. You have looked at asbestosis mortality and smoking, in other publications?

A. No. Usually we don't know what the smoking is in a mortality study, and we do know for a subgroup of the men at the London factory because a special undertaking was carried out there which led to the paper we were discussing yesterday, number two, tab two.

25 But apart from that, in mortality studies one doesn't usually know the smoking habits because the mortality studies are cohorts which are constructed from historical data, which is from data in factory record systems, and these historical records don't record smoking. They only record things such as job and identity details.

30 Q. I'm just...do you recall a paper that you

Q. (cont'd.) wrote that you wrote with Mr. Rossiter, and presented in 1978...

A. Yes.

Q. ...wrote in 1978, on the Interaction of Asbestos Exposure and Smoking on Respiratory Health?

A. Yes, what number is that?

Q. I'm not sure it's in. I've got a copy of it though, if you would like to look at it.

A. Yes. I think I've probably got a copy.

MR. HARDY: Maybe we should make that the next exhibit.

THE WITNESS: A. Yes, I have a copy with me.

MR. HARDY: Q. Good.

MR. LASKIN: Exhibit fifteen.

EXHIBIT # 15: The abovementioned document was then produced and marked.

THE FOREGOING WAS PREPARED
FROM THE TAPED RECORDINGS
OF THE INQUIRY PROCEEDINGS

Edwina Macht
EDWINA MACHT

...to page 26

MR. HARDY: Q. As I read this paper, you consider, among other issues, the relationship between smoking and asbestosis?

THE WITNESS: A. Yes.

Q. And is perhaps -- and this deals with some data from a different group from the Rochdale cohort; isn't that correct?

A. Well, the Rochdale group is included, and also a group which Mr. Rossiter was studying, of dockyard workers.

Q. I gather it's Mr. Rossiter's half of the paper that contributed the royal dockyard data.

Do you recall whether the same finding of relationship between smoking and asbestosis was true with this other cohort as in your Rochdale cohort?

A. Yes; I think it was a fairly similar finding, but if we look at the abstract, it was only in people who were heavily exposed and smoke that the prevalence rates were raised.

Q. And I believe, if we look, perhaps, at table 4, on page 201, which is data from the Royal Naval dockyards, we can see the effect of smoking -- or, at least, bear on prevalence of opacities.

A. Yes; that's right.

Q. So this is the second set of data which, like your data at Rochdale, shows greater risk, not only for lung cancer, but also for asbestosis, among smokers?

A. That's right; yes. The dockyard data isn't as clear-cut as the Rochdale data.

Q. But tends in the same direction?

A. Yes.

Q. Yesterday, you discussed not only dose-response relationships that you found in the various studies you've done

Q. (cont'd) with lung cancer and asbestosis, but also indicated that you had found some indications in the dose-response relationship for mesothelioma?

A. Yes.

Q. And I gather you found that in the East London factory data.

A. Yes.

Q. And I guess -- we looked at the table yesterday ---

A. Yes.

Q. And I gather that, in the gas-mask factory study, which is number 13, which you worked the same -- a similar dose-response relationship where mesothelioma was found?

A. Yes. But when we say similar, in the gas-mask workers, the only measure of dose was length of exposure.

Q. And I think maybe we could look at table 5, number 13 in your articles, and perhaps we can see clearly what you're talking about; it's on page 646.

A. Yes.

Q. Maybe if you could just explain to us that table, please.

A. Table 5 shows the women who worked in this factory, assembling gas masks, sub-divided by the length of exposure, the time that they worked there. And all this work was going on at the beginning of the war, or just before the war; that's round about 1940 to 1943, I think it was.

Nobody worked on it for more than about five years, at the most, and the thirty -- the final group, thirty months plus is probably up to about five years. If we look back, I'm sure we'll find the exact figure, but we're not talking about people who may have been exposed for twenty years. They were all exposed for quite a short time; thirty-five years

A. (cont'd) before this paper was written.

5 So that the -- we haven't got any problems of differential weightings of the type we were discussing earlier, because all this exposure was thirty-five years ago.

10 We divided them by months; less than one month, those who worked from one to three, et cetera. We've got the number of women, and then we've got, in the next column, the number of those who developed a mesothelioma, to date, and with the percentage given in brackets.

15 The mesotheliomas didn't occur in anybody who'd been exposed for less than five months. And I gave, in my evidence yesterday -- I summarized this statement by saying there were none out of three hundred and fourteen in one group, compared with sixteen out of four hundred and twelve ---

15 Q. In the longer exposed group?

A. That's right -- which I used to illustrate that there was a dose-response relationship.

20 Now, in the actual paper, we looked at it in more detail than that, and we actually fitted a dose-response relationship through the data; instead of just summarizing it by two groups, we kept it in the seven groups that we have in the table and fitted a dose-response relationship. And, in fact, a linear relationship fitted these data very well.

25 Q. Have you done any work, or are you aware of any work, which attempts to determine whether there's a dose-response relationship for mesothelioma in animals -- studies?

30 A. Yes. There's an experiment that we did at the pneumoconiosis, which was summarized in one of the extra papers that we were talking about yesterday. I haven't got the reference with me because they're away being photocopied. It was from the British Journal of Cancer, 1973, where we -- it'd be by Wagner, et al. ---

Q. That's number 17 on the extra references, just for the record?

A. Yes; that is correct.

MR. LASKIN: Perhaps if we could make this part of the record -- and I don't mean to interrupt Mr. Hardy -- we should just mark this extra reference page as an exhibit, so we know what we're talking about. It will be Exhibit 16.

EXHIBIT # 16: The above-mentioned document was then produced and marked.

THE WITNESS: And in that paper, an experiment is described in which rats were inoculated with asbestos at doses ranging from a half a milligram to eight milligrams, in steps of doubling; there was half, one, two, four, and eight milligrams.

And the number of mesotheliomas increased with dose. And we fitted a dose-response relationship through those data and found a linear relationship was consistent with the data, although it was quite a small experiment, and other dose-response relationships were possible. But a linear one was within the range of those possible.

MR. HARDY: Q. You say other dose-response relationships were possible. What other possibilities were there?

THE WITNESS: A. The dose-response relationship we were fitting was one of the form that the incidence of mesotheliomas was proportional to the dose raised to some power, and we then estimated that power.

And the estimate of that power had limits which went below one and above one -- you know, without having the paper -- I've forgotten the details, but maybe it went from, say, point 6 to one point nine. We can look it up.

So that means there was a possibility that there

A. (cont'd) could have been a dose-response relationship which curved upwards.

5 Q. One final subject I guess I'd like to ask a few questions, Mr. Berry. Mr. Laskin, yesterday, spent some time discussing with you a presentation you made, entitled "Hygiene Standards - Theory and Application," which is number 4 of the articles in your collection.

10 And you discussed with him a number of health-related factors that would go into determining, in your view, an appropriate control standard for a substance.

And you also discuss in the article, but I don't believe it was discussed too much yesterday -- at least, make reference to some non-health-related factors that go into determining an appropriate standard.

15 A. Yes.

Q. You know the one I might be referring?

20 A. Well, I can't find it right now, but I guess we're talking about the benefits of using asbestos; in other words, this is all based on the assumption, which is given on page 146, right at the beginning. I say "assuming that asbestos is an essential material."

Of course, if we weren't assuming that, then the whole problem would be different. If we're assuming we need asbestos because of its benefits, then that is one of the non-health criteria.

25 Q. And so that I gather there has to be balancing of some sort done in standard-setting?

A. Yes.

MR. HARDY: I don't think I have any further questions right now, Mr. Chairman.

30 DR. DUPRE: Thank you, counsel.

Who is next in the line-up?

MR. McNAMEE: I believe I am.

DR. DUPRE: Mr. McNamee, if you please.

CROSS-EXAMINATION BY MR. McNAMEE:

MR. McNAMEE: I put out too many questions, and maybe, while I was out of the room, Tim might have asked a couple, so, if I repeat some of his questions, please stop me.

Q. I understand that Miss Jolie's employment with the Simpson's Commission, or whatever, has recommended certain proposals for standards, and are you in agreement with the proposed standards, or do you propose something lower? Would you recommend something lower than the proposed standards?

THE WITNESS: A. Well, I think -- I wouldn't personally recommend any standard, because I think standards, as we were discussing just a moment ago, have to be drawn up to balance advantages and risks; a lot of social judgement goes into how to do that.

It has to take account of whether or not the substitutes which some people say are adequate, whether, in fact, they are adequate, and I think these decisions can only be made by a committee, which has experts in all the different fields considered.

And I did say something along those lines in this paper, tab 4, which we were discussing a moment ago. But I think the Simpson Committee have produced a good document, and reached conclusions which seem, to me, to be very sensible.

I think I'm correct in saying that the standards which they propose, they only propose as a step at this time; I think that they propose action in the future to bring them down further. In other words, they regard their proposals as a first step, or the next step of a continuing process.

Q. Thank you.

In your study -- I believe it's tab number 6,

5 Q. (cont'd) "Patterns of Mortality in Asbestos
Factory Workers in London," there are mortality experience
tables, comparing the female and the male, and, quite apart from
the question of susceptibility, I notice that -- at least, I
thought that the incidence of cancer was somewhat higher in the
cohort of women, and I'm wondering if that might be accounted
for by the fact that the nine hundred and twenty-two women all
entered the work force between January, 1936, and December, '42
10 -- it would appear to me that they were probably, overall,
exposed to heavier doses of various forms of asbestos than the
male cohort, which covers a period from April 1st, 1933, to
March 31st, '64.

Do you have any comments on that?

15 A. Well, there's two points. When you say the
women had a higher incidence, I don't think that's ---

Q. Sorry; in "Severe," over two years, if you
look at table 6 on page 58, as compared with the men who are set
out in table 1 on page 54. Severe exposure over two years, the
ratios for the women seem to be much higher for cancers.

20 A. Well, yes. Of the women, there were twenty-
one lung cancer deaths, compared with, out of seventy-eight
deaths altogether, so that is about one death in four. In the
men, in table 1 on page 54, there are fifty-six out of one-
seventy-six; that is one in three. In fact, the men have a
higher incidence.

25 But if we then compare them with expectation,
which is, I think, the point you're making, the ratio of
observed to expected is much greater in the women; but we end
up with a slightly lower overall incidence, because the lung
cancer rate in women is so much lower than for men. So we've
got a higher factor.

30 Now, you're asking, could that be due to higher

5 A. (cont'd) exposure. I don't really know the answer to that, because both the men and the women were doing severe jobs; but there are traditional jobs which were done by women, and others that are traditionally done by men. So, on the whole, they would be doing different jobs; they were both classified as severe.

There could have been different dust levels, but we have no information on that.

10 Q. Well, what those figures (1936 and 1942) suggest to me is that, say, by 1960, most of those women may very well have left the work force, at a time when better standards were being introduced; as you point out on page 53, there was a betterment of the working standards.

15 And it just appeared to me that the women cohort may very well have spent a greater period in the time when standards were lower.

20 A. Yes; there was certainly, as you say there, in the 1940's where some of the men arrived later. But a lot of the experience of men would be drawn from those people who were there at the same time, but I don't have an exact comparison with me.

25 But we did look at time of starting for men, and compared men who started before and after 1955; and we probably didn't mention that in the paper because we didn't get any definite conclusions from that comparison, because those who had started since 1955, we'd only just got, at the time we wrote this paper, past the twenty years' point of latency, and there weren't many lung cancer deaths that had occurred up to that point.

30 So that's why I'm reasonably confident that the men -- most of the men lung cancer cases would have been exposed certainly before 1955. Any clean-ups, say, that

A. (cont'd) happened in the 1960's would not be relevant to the point that you're making.

5 Q. For the women, though, you did have a long period of follow-up; their group is better documented, really, than the men. I mean, another ten years, you'll practically have your study -- a complete study on the women in that particular factory?

A. Yes.

10 Q. You mention, in -- well, over the period of years, the methods of testing have changed, and there's been some discussion about correlating results by one method as opposed to a previous method.

15 And I just noticed in one of your papers -- I believe tab number 7, "Dose-Response Relationships for Asbestos-Related Disease," at page 192, you do talk about simultaneous sampling with two methods (with the impinger and fibre counting) in a New Orleans plant.

20 And I just want to -- has there been much parallel sampling, with a view to using parallel sampling, to test the correlations that people have used. Do you understand the question?

A. Yes. I just haven't quite found this part on page ...?

Q. Sorry; page 192.

A. Oh, yes, I see.

25 Q. Under "Other Studies."

A. Yes.

Q. And, really, what I wanted to know, whether there had been much use of parallel sampling just to test some of these correlations, or are these correlations often just a matter of guesswork?

30 A. Oh, I think in some places there's been quite

5 A. (cont'd) a lot of studies. I think Dr. Gibbs has done a lot of studies. No doubt he'll tell you about those. But it's not a subject I've got any direct knowledge of; in this part, I am referring to other people's work.

Q. Do you yourself use any correlations in your work -- I gather that really you didn't try to correlate.

A. No.

10 Q. Is there any validity in - well, taking all -- there are a number of experts, like yourself, who have done studies; some in plants throughout the world -- is there any validity in trying to bring all these studies together, and to derive any additional information that might result as sort of an accumulative basis, or **does each** study more or less have to stand on its own?

15 A. No; I think the studies have to be brought together, to see what sort of patterns emerge; where there's agreement and, equally, what sort of discrepancies emerge.

20 For example, the point I was making about the association between mesothelioma and amphibole can't really be made very strongly from one study alone. There's no single study which -- from which one can draw that conclusion, without risk of error.

25 But when one puts all the studies together -- and you'll find a good discussion of this in the Simpson report by -- the part by Acheson and Gardner -- when you put all this information together, quite a clear picture emerges; though I know there are one or two people who still disagree with this point.

30 The slide I put up yesterday, towards the end, was an attempt to compare the studies that have been done in different areas where there's a dose-response relationship, and see to what extent these agree. And, as we saw, there was one study

5 A. (cont'd) which doesn't fit in with the others at all, and obviously one would like to know why that is; but, at present, I don't think we do.

10 Q. Just to develop that a little bit further -- it's my last question -- say, an individual study might show, on a limited number of observations, a ratio of, say, three cases observed to two expected, which probably wouldn't be statistically important in one case, but if ten studies showed ratios of three to two and four to two, the total number might be statistically important. Is that correct?

A. Yes.

MR. HARDY: Thank you; those are my questions.

15 DR. DUPRE: Miss Jolie, would you wish to take a five-minute break before -- at this point -- before proceeding?

[No audible reply.]

INQUIRY RECESSED

.....

INQUIRY RESUMED

20 DR. DUPRE: Miss Jolie, do you wish to proceed, please.

CROSS-EXAMINATION BY MISS JOLIE:

25 MISS JOLIE: Q. Earlier, my colleague from the government was pursuing the issue of a number of studies, and I think you were discussing the fact that the Dement study seemed to be out of line with a number of the other studies.

However, the Selikoff study -- it's not really out of line with some of the Selikoff studies; is that correct?

30 THE WITNESS: A. Well, I was only considering studies that were of chrysotile, or mainly chrysotile.

Q. Ah; right.

5 A. Actually, Selikoff gives very little dose data, in any of his papers; but I know people have constructed a dose-response relationship from something in one of Selikoff's papers, and it is ---

Q. Dr. Enterline just suggested a dose-response relationship from Dr. Selikoff's material that was more overt to theirs as well.

A. Yes.

10 Q. I'd like to pursue a little bit of the discussion yesterday about the under-diagnosis of disease, and, in terms of mesothelioma, my understanding, in some of the Newhouse studies, is that there's a fairly significant under-estimation, or under-diagnosis, of mesothelioma. Is that correct?

15 A. Well, I mean, the short answer to that is that -- well, no; perhaps I'd better not give a short answer. I don't think we ever say that in any of the papers, do we, that there's an under-diagnosis?

20 But the diagnosis is based on collecting post-mortem material, where available, collecting hospital notes, and that sort of thing, and having these reviewed and, in particular, having the histology reviewed. And that's how a lot of the mesotheliomas have come to light.

25 Now, there are people who die with no post mortem, and particularly for the early years, there were people who may have had a post mortem but the material's been thrown away, because it was so long in the past. That only, of course, related to her early work, where she was going backwards in time. With the updates, we're only looking at recent deaths.

30 And I think, yesterday, I said that the post-mortem rate might be about fifty per cent, but I checked afterwards, in one of the papers (which is number -- I can't see it right now -- ah, yes; number -- no) -- no; actually, I don't

5 A. (cont'd) think -- I think it's one that's not on the record. But it was a paper from the British Journal of Industrial Medicine in 1972, one on the mortality of the women, and, there, out of a hundred and forty deaths, there were post-mortem reports on fifty-one; so, in that case, it wasn't as high as I perhaps indicated, at fifty per cent.

10 And, of course, for those where there wasn't a post mortem, and where they died of, say, lung cancer, then it's possible that some of those were mesothelioma.

Q. And is it possible that some of the gastrointestinal cancers were also misdiagnoses, or, rather, cancers like pancreatic cancers might be misdiagnosed?

15 I understand Dr. Newhouse also found that was one of the most common misdiagnoses, was pancreatic cancer, for peritoneal mesothelioma.

A. Well, that sort of misdiagnosis is possible, yes.

20 Q. We had testimony before the Commission last week, by the insulation workers, both here and in the United States, that, in fact, in the study that they've done with Irving Selikoff, that one-third of the death certificates, when they went back to investigate, in fact, were incorrect.

Would that be your experience, too, do you think, in England, or -- well, obviously, you have more post mortems, perhaps, than we do here?

25 Do you see that as a major problem, if one doesn't investigate the death certificates?

A. Certainly, it's a problem with mesotheliomas, and particularly more so for periods twenty years or more ago. And I know other misdiagnoses have occurred, but I can't recall the full details of these and what the percentages are.

30 There was a paper by Dr. Newhouse and Dr. Wagner,

5 A. (cont'd) and it's Dr. Wagner who looks at all Dr. Newhouse's material -- I've forgotten what year, but it was quite a while ago; round about 1969, I think -- where they gave details of the comparison of the death certificates and Dr. Wagner's post-mortem opinions for the men at the London factory.

And I can't recall the details, but I think mesothelioma was the main problem. And there were other misdiagnoses, but I don't think they were too serious in total.

10 And I know the studies of Selikoff's group, they often -- in recent years, they've given two columns of observed; they've given death certificate causes and what they call "best evidence." They usually have a column with "B.E." above it -- "D.C." and "B.E."

15 Again, I'm surprised that the discrepancy's as high as one in three, but, no doubt, if that's what they told you, it is correct; but there may well have been some misdiagnoses in both directions, so that, in the group as a whole, the discrepancy might not have been as big.

20 Q. I just wanted to ask you a question, because I don't know what this term is -- and it's in number 9, "Mesothelioma in the United Kingdom," on page 197, and it's "mesodermomas." You indicate that there were a number of mesodermomas connected with asbestos exposure, and that "Our cases would suggest that they are" asbestos-associated. And I just had never heard that term before and wondered what they were.

25 A. I'm not sure exactly; I know this is -- Dr. Jones, who was the first author of this paper, was very keen on this, but I couldn't tell you, medically, what a mesodermoma is.

30 But I think it's -- I think it's something -- I mean, the "meso" implies it's something fairly similar to a mesothelioma, I think; but I think you'd better get a medical

A. (cont'd.) opinion on that.

Q. I just had never heard the term before; I was interested.

A. The point that was being made, that there might be other tumors that were related to asbestos, and there were just three of these.

Q. When we were discussing the whole weighting of dust exposure in the early years, it has implications I consider for compensation purposes, and that is that, in our jurisdiction, we will not compensate people unless they have continuous and repetitive exposures for at least ten years; and a number of compensation cases are denied, based on that continuous and repetitive exposures.

But given that weighting, do you feel that that's a very stringent criteria to use? It's used -- excuse me -- in almost all the cancer; it's used for mesothelioma, it's used for lung cancer, it's used for gastro ... No; gastro-intestinal cancer is just generally a long-term, repetitive and continuous exposure.

But that's the criteria on which a number of our cases are refused, and it seems to me that's ...

A. Well, I would think, certainly, for mesothelioma that those criteria are very stringent, because mesothelioma, although there are cases that occur which can't be traced to any asbestos exposure, it is a very rare disease, and it is known most of them are related to asbestos exposure.

So if somebody has worked in an asbestos factory for a year or two, then mesotheliomas have been shown to occur; such as in some of the slides I showed yesterday, where there had been quite a lot of mesotheliomas with less than two years' exposure; and, again, in the gas-mask group.

Q. In the case of lung cancer, if you had, in

5 Q. (cont'd.) the very early years of exposure, very high levels of exposure, or even high, intermittent levels of exposure that we discussed, the possibility of those being more dangerous, is that not also, therefore, a fairly stringent criteria, perhaps, for lung cancer as well?

10 A. Yes. Again, we could refer to the work at the London factory, at tab number 6; we could look at table 1 on page 54, for the severe group, there've been thirty-one lung cancers - well, six of those are mesothelioma, so, if we take those off, there've been twenty-five, and twelve point eight were expected. So there is evidence of an increase in mortality due to lung cancer, even taking pleural mesotheliomas out, with -- well, in this case, less than two years.

15 Q. I'd like to ask a few questions on fibre type, if I may, and almost all of your comments yesterday dealt with mesothelioma. You did refer to Enterline's work as far as lung cancer was concerned.

20 One of the quotes that interested me, and I wondered -- it was from your colleague Julian Porter -- Peto; sorry -- jurisdictional slip; sorry. [Laughter.] He's a lawyer in Toronto.

25 But Julian Peto indicated that perhaps peritoneal mesothelioma was due to crocidolite asbestos; whereas, chrysotile asbestos may lead to pleural mesothelioma. That was based on his study of the Turner-Newall; it was a quote from his study.

And I wondered, does that fit in with some of the evidence that you've come across, or ---

30 A. Well, of course, the first thing to say is, it's based on the Rochdale factory, where crocidolite was used over a long period of time. There wasn't as much; chrysotile was the predominant fibre, but there was crocidolite throughout

A. (cont'd.) the factory over a long period of time.

5 And I think I mentioned yesterday that we had some data, which is, as yet, unpublished, on lung contents of post-mortem cases from the Rochdale factory, and they do support that there was crocidolite there. And there's quite a high amount of crocidolite in the lungs of some of these Rochdale cases, as much as in the mesotheliomas in the 1976 study, which we were
10 looking at earlier, or almost as much.

So we mustn't think -- regard the Rochdale factory as pure chrysotile; that's the first point.

15 And then I think there have been studies elsewhere in the world where the exposure is undoubtedly to crocidolite, where only pleural mesotheliomas have been observed. And I'm thinking particularly of the Australian study of those people involved at Witloom, where there'd been quite a number of mesotheliomas (twenty or more, I think), and all of them had been pleural.

20 Q. The experience in Quebec, however, with the chrysotile exposures, seems to me most of their mesotheliomas were pleural; is that correct? The McDonald study.

A. I don't recall offhand. It wouldn't surprise me.

25 Q. In any event, chrysotile does lead to mesothelioma, albeit at a lower rate?

A. Yes.

Q. The other paper I'd like to refer to, in terms of fibre type, is number 18 in the new list, and that was your study with Wagner and Skidmore on the effects of the inhalation of asbestos in rats.

A. Yes.

30 Q. And in that particular study, you found that,

Q. (cont'd.) in fact, chrysotile -- the deposits of chrysotile fibres were considerably lower than the deposits of crocidolite fibre.

A. Yes.

Q. But, in fact, the carcinogeneity and the fibrogenicity of the fibres were the same in the rats.

A. Yes; very similar.

Q. Could that lead to a conclusion that, in fact, they're more fibrogenic -- chrysotile could be, in fact, considered more fibrogenic and carcinogenic in rats at these lower deposits, then?

A. Well, I can see -- I can see your line of argument there, yes, and it's, you know, one I'm not sure of, because we've contrasted the inhalation with the injection studies.

Now, we know, with injection, that the whole dose gets there. So if we compare the paper you've just referred to with paper number 17 -- papers 17 and 18 use the same materials; one by injection, the other by inhalation -- then, pursuing -- and I think we discuss this in one of the papers -- but pursuing the point you made, if, after inhalation, there's a similar effect, we know that much less chrysotile seemed to get into the lungs of the rats, then we would expect that, after injection, the effect of chrysotile would be -- show up much more than the crocidolite. This didn't occur. And, you know, I don't really fully understand why this is.

Q. The Simpson report, in fact, came to the conclusion that levels -- that, even among the amphiboles, levels should be different, or standards should be set for different levels.

A. Yes.

Q. Amosite, presumably, at point five, and

Q. (cont'd.) crocidolite at point two; or considering a ban on crocidolite.

5 Do you feel that the scientific evidence supports that as well?

A. Well, on the whole, I think it's a reasonable thing to do from the scientific evidence; but I think, as they mention, there isn't a great -- all that much evidence, but what there is suggests that amosite isn't quite as dangerous as crocidolite.

10 Q. I'd like to go back to the smoking discussion we had this morning, and that was that you indicated that, in fact, Selikoff's -- Hammond and Selikoff, and perhaps Sideman's most recent figures show the relationship more clearly.

15 What it also shows more clearly is that, in fact, non-smokers are now producing lung cancer.

A. Yes.

20 Q. And Dr. Enterline suggested to us last week that perhaps the reasons that we didn't see the lung cancers before was because, in fact, smoking probably shortens the latency period of the effect of asbestos.

The combination is not only multiplicative in its effect, but it probably shortens the latency to time to tumor?

25 A. I see; yes. I mean, that is one possible explanation, but another is that, in the early study, the first paper of Selikoff, where I don't think there have been any lung cancers in non-smokers, the expectation was very low. I think -- I've forgotten the figures, but the expectation was so low that even if there'd been -- even with a sixfold excess, or fivefold excess, it was not surprising to find zero cases.

30 And, of course, this is a point brought out by Dahl in a paper in 1971, when he analyzed the original Selikoff smoking data and suggested a multiplicative model which fitted.

5 A. (cont'd.) And so, from that, it follows that the explanation could just be, because there hadn't been enough time elapsed -- not to achieve a latency, but not enough time to achieve a big enough expectation of cases.

10 Q. Dr. Enterline also presented to us information from Radford, at the University of Pittsburgh, on uranium mining, and, in fact, the uranium miners had the same experience but, as time went on, the effect of smoking seemed to be cancelled out and the lung cancer rates between the non-smoking and the smoking were essentially the same after a long period of time of following the people in Colorado.

15 And Dr. Enterline suggested that perhaps what we're seeing here may be the same thing; that, after a long period of time, we may, in fact, see that non-smoking asbestos workers have an equal risk of getting lung cancer.

A. I see; yes. I haven't heard that put forward before, that idea. I mean --

Q. Is there -- oh, sorry.

20 A. I was going to say, what does he mean by "a long period"? I'm trying to recall what the period of follow-up was in the Hammond, et al. I mean, they had quite a long period of observation, I think. I was thinking of something quite a lot longer.

If it got too long, of course, it would only be of academic importance.

25 Q. Is there a relationship between smoking and mesothelioma?

A. No; no such relationship has ever been demonstrated, as far as I'm aware.

30 Q. So that encouraging people not to smoke will not alter mortality by mesothelioma?

A. No.

5 Q. I'd like to go to the dose-response relationships, and your study number 7, on page number 192. We have discussed this, but I just want to understand clearly exactly what you've been saying.

And that is, towards the bottom of the page, or at the bottom of the first paragraph, under "Other Studies," you say:

10 "It was not possible to convert this exposure to fiber-years ... because there was a low correlation between impinger and fiber counts."

You're dealing with McDonald's study; an earlier study of McDonald's.

Is that still true?

15 A. Well, I know McDonald and his colleagues are working on this, and they gave some preliminary results at the Lyon meeting which were published last year, and so I don't think it is quite true, no.

20 They've now done work which is enabling them to do more than they felt they could do when they wrote this New York paper.

Q. But, still, it's based on a lot of assumptions, in that the parallel measuring now is assuming that there's always constant dust clouds, and that kind of thing, in the past. Is that correct? I think John Laskin dealt with that yesterday.

25 A. I think that is probably true; but you can confirm that with Corbett McDonald and Graham Gibbs.

30 Q. Given that perhaps we can make those leaps, those conversions, do you feel that we should be using studies done on chrysotile miners and millers to set standards for workers in production plants, in textile plants, in friction products, et cetera?

5 A. Well, we did touch on this yesterday, didn't we, when we were discussing whether the differences between various studies might be due to the type of process.

10 And I think there is some evidence, particularly with the Dement study; although one can raise question marks about that, it does look to be quite different. And that is a textile operation. The Peto study is a textile operation. And they both -- although they are quite different, they're both higher than friction materials, and they're higher than mining and milling. So I think there's reasonable evidence to suggest that.

15 Using mining and milling data, applying it to other industries might not be the best thing to do.

20 Q. The other thing that we were talking about, standards, and I know we can't pin you down on standards, but you've suggested a committee of experts should consider this.

Should it not include people who are actually going to be taking the risks?

A. Yes.

25 Q. I mean, the determination of the socially acceptable standard should certainly include people who are going to take the risk.

A. Oh, certainly; yes.

30 Q. I guess I was -- perhaps we are experts, so ...

25 The other -- I just have two more questions, and that is that you talked about control limits, and you talked about substitution as part of a control limit.

A. Yes.

30 Q. Now, obviously, we would not want to substitute another hazardous product for asbestos, but if we were to find alternatives that were safe (safe in terms of being

5 Q. (cont'd.) properly tested before we use them), would it be a good practice, in fact, to pursue substitution of asbestos for those products that we're able to find alternatives?

A. Yes. I mean, with the caveats you've put on it, I don't see how one could disagree with that.

10 Q. And the last thing I'd like to ask you was in your paper number, which was in your discussion summary -- and I realize that what you're doing there is summarizing the discussion at a conference, and some of this may not be your opinion -- but the last paragraph, on page 863, you were summing up the dis... -- or you were summing up the chairman's summation, and talking about the concept of linear dose-response relationship, and you ended your statement with: "Acceptance of this is a major step forward."

15 And I just wondered if you could elaborate on that statement.

20 A. Yes. What the chairman of this discussion had in mind -- and it doesn't say here who the chairman was, but it was actually a Dr. Westerholm from, I think it was, Sweden -- what he had in mind was that, if everybody could agree that a linear dose-response relationship was valid, then one could -- it gave one a basis for extrapolating from high doses to low doses, without worrying about things such as a threshold concept, which we were talking about yesterday, or the elbow-type effect, which was mentioned.

25 And I think what it meant, it was a major -- it would be a major step forward because a lot of the discussion has been over things such as whether a threshold is a reasonable assumption or not.

30 Q. In the scientific community in Europe, is there general acceptance of the linear dose-response relationship?

5 A. Well, I wouldn't like to answer that too definitely. I mean, I know that most people who've been handling data from studies such as this accept it, but I think quite a lot of medical people dispute it.

MISS JOLIE: Thank you very much, Mr. Berry.

DR. DUPRE: Mr. Bazin?

MR. BAZIN: There's still a few questions, Mr.

Chairman.

10 CROSS- First of all, I would simply like to, by way of a question -- Miss Jolie -- and I don't want to engage a debate here, but she has stated, on two occasions, that one-third of the death certificates have been -- had to be corrected. I have not heard testimony to that effect here.

15 You know, I just want to make that mise au point. As far as I am concerned, I haven't heard that such an occurrence took place, and I don't want to engage into a debate on this one.

20 DR. MUSTARD: Mr. Counsel, that will be as part of our Phase I hearing, the documentation that Miss Jolie was referring to.

MISS JOLIE: Yes.

DR. DUPRE: I believe, Miss Jolie, you were referring to the appearance, early in June, of the representative from the insulation union in Washington.

25 MISS JOLIE: Right. And Dr. Uffen pursued that question with Dr. Enterline on ---

MR. BAZIN: It was raised by way of questions.

30 DR. DUPRE: I'm just saying, for your reference, counsel, you may wish -- or we should get to you, if you don't have it now, that brief that's a matter of public record. And, of course, the transcript from that meeting will be available for you to examine.

5 DR. UFFEN: Could I also make the comment, my recollection was that the gentleman who spoke to this volunteered to supply us with the evidence from Selikoff's data; and I will watch to see that we get it.

CROSS-EXAMINATION BY MR. BAZIN:

10 Q. Dr. Berry, to pursue, briefly, on the question of the acceptability of risk which you deal with in your paper, tab number 4, first of all, if I understand, Dr. Berry, you are part of the group there, a unit -- pneumoconiosis unit in a hospital in England; is that correct?

A. In Wales.

15 Q. In Wales. And, as such, you have been doing the statistical analysis which this group had to do on various minerals; not only asbestos?

A. I have done work with other materials, yes.

Q. Have you done any work on lead, for instance?

A. No; I've done no work on lead.

Q. On copper?

A. No.

20 Q. Uranium?

A. No.

Q. Nickel?

A. No.

Q. Gold?

A. No.

25 Q. Silver?

A. No.

Q. But you have done work on other minerals, I imagine, that are found in the United Kingdom?

A. Well, I've done work on other dusts; for example, I've done work on cotton workers.

30 Q. On cotton workers?

A. Yes.

Q. Cotton workers where asbestos was being used?

A. No; straight cotton.

Q. Could you tell us what kind of dust -- what the description of the dust particle in the cotton industry where asbestos is not used; what's to be found there?

A. Uh -- no; I'd prefer not to answer that one. I don't really know the answer offhand, in the terms that you want it.

Q. Coming back on the question of risk, you mentioned that it was, as far as you were concerned, a balancing act which had to take into account many factors; is that correct?

A. Had to take into account ...?

Q. Many factors.

A. Many factors; yes.

Q. And one of these factors is substitutes?

A. Yes.

Q. Are you satisfied that there has been proper analysis done of substitutes, insofar as asbestos is concerned?

A. Mmm -- well, the -- the main substitute that I'm aware of is glass wool, and that's fibre; and things like rock wool for heat insulation purposes.

And these materials are, at present, being investigated fairly vigorously; animal experiments are being done. And the main conclusion from these is that, if one controls the fibre size, so that there are -- most of the fibres are thick -- then, from animal experiments, that would indicate that they were certainly a lot safer than asbestos.

And, of course, with asbestos, one can't control the diameter to any significant extent, because that's a property of the mineral as it occurs in the ground.

5 A. (cont'd.) There's also epidemiology being done on glass-fibre workers, but this hasn't got too far at the moment; and one reason is, because there hasn't been a sufficiently long follow-up.

Q. Are you talking about the same length of follow-up there that you are talking about with the asbestos ongoing examinations -- ongoing ---

10 A. Yes; I'm talking about that if glass fibre was going to produce a carcinogenic effect, then it would not be detectable in man until at least there'd been a twenty-year follow-up.

And I don't -- there's very little data; I'm not sure if there's any at all, but there's very little data where that has been done.

15 Q. On the question of dust particles and substitutes, could I refer you to your paper, tab 11, "The Comparative Effects of Three Chrysotiles ..." on page 364.

I take it that these were, quote/unquote, pure chrysotile fibres that were injected or forced by way of inhalation in the rats?

20 A. Uh -- well, it does say, on page 364, that none of the three samples was pure.

Q. There were other things ---

A. No; there were contaminants. Yes.

Q. Contaminants?

25 A. Yes. Perhaps some of these are listed; this is in the first paragraph on page 364.

Q. I wanted to have your comments on that precisely.

30 Are you satisfied that, from a statistical point of view, factors have been -- or that co-factors have been taken into account when examining what asbestos has caused at

Q. (cont'd.) various doses for various people during various lengths of time?

5 A. Are you thinking here of comparing different studies?

Q. No; I'm thinking of comparing what may be in the dust in a textile factory with what may be in the dust in the mining and the milling part of the industry, and the influence that co-factors may have on the health of those employees.

10 A. Right. So they were contrasting textiles with mining. I take it the point you're getting at is that there might be a different distribution of other co-factors in different working environments ---

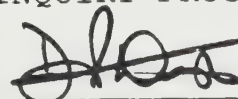
Q. Yes.

A. --- which might influence the final outcome.

15 Yes; I would think one could put that forward as a possible reason for some of the differences we were talking about, but I don't have any data on, you know, whether the percentage, contaminants, to what extent they do differ between these different work operations. That would have to be done.

20 Q. Leading into the question of a possible asbestosis diagnosis, which is discussed particularly in your tab number 8, could it be possible that some of the signs that lead to the diagnosis of possible asbestosis may come from some of these co-factors?

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THE FOREGOING WAS PREPARED
FROM THE TAPED RECORDINGS
OF THE INQUIRY PROCEEDINGS



DEREK WEST

5 A. Yes, it could be, as we discussed somewhere in this paper, there's not only that point, but I suppose it's the same point so we may have false positives...that is, people who have got various signs which lead one to believe they've got asbestosis, whereas in fact they may not have asbestosis, which I think I'm right in saying and you can no doubt get a medical man to confirm or refute this. But in life there are cases where it is difficult to say whether the symptoms are due to asbestosis or not without a post mortem, then it's easier.

10 Q. Could that explain a fifty percent result in the possible asbestosis? I understand that of those who were diagnosed as possible, fifty percent eventually...fifty or sixty percent eventually had asbestosis?

A. Were certified.

15 Q. Yes, I'm sorry, were certified.

A. Which is another sort of clinical judgement, that one has to have more..be in a more disabled state to pass through that diagnosis.

Q. Yes.

20 A. Yes, I think that's possible and I'm pretty sure we discussed that in the paper. I can't quite find the page.

Q. Are you able to express a judgement as to the satisfaction level of the fifty percent result in the diagnostic...?

25 A. Well, we are not going as far as saying that, because only sixty percent were certified within the period of followup we had. The other forty percent were false positives and some of them might be certified at a later date. So the misdiagnosis is not as high as fifty percent.

30 Q. Going back in the diagnosis, there is a number of references to crepitations.

A. Yes.

Q. My understanding of what these may be in laymen's understanding, they are a form of rales, crepitations?

A. Yes.

Q. They are not the same thing? There are different sorts of rales?

A. Yes, I'm not too sure of this. Some people say they are the same thing, and some people say there is some sort of difference. Certainly they are very similar, in my understanding.

Q. Crepitations, according to some, may be a form of rales?

A. Yes.

Q. Are you aware of studies which lead to the conclusion that basically everyone has creptiations of some sort?

A. No. No, I wasn't aware of a study where data had been put forward like that, but we were aware in doing this study that several of the signs, including radiological changes, could occur in the absence of dust exposure of any sort.

Q. Talking about the absence of dust exposure and the question of elimination, which you deal with more extensively in your paper number seven, page 187 at the bottom... I don't want to go through the whole description once more, but on the question of the elimination, would you agree that an investigation of a population in a one industry...being an asbestos mining area...an investigation of the general population not working in the mine where known levels of dust exposure exist in the city, and where there have been no cases, no known cases of asbestosis or lung cancer more than the standard, that that could give you results of the elimination process of asbestos in those individuals?

A. You mean post mortem studies?

Q. Yes.

5 A. Of how much they had in their lungs? I wouldn't have thought that would be a very sensitive study because you are talking about very low exposure. You are talking about nonoccupationally-exposed people, and therefore I take it you are assuming that there is some general environmental exposure which you can measure?

Q. Yes.

10 A. That this would be so low, the amount of dust would be low relative to the occupationally exposed, that it would be fairly insensitive compared with doing a study on people who are actually working in an occupational environment where you also had measurements.

15 Q. But where you can't say precisely what the elimination process is?

20 A. That's correct. One can't say that because the data necessary have never been obtained. One could envisage that they could be obtained, and I would think that if that was the case it would be better done on a group who had higher exposures and then been withdrawn from exposure because they have left work.

25 In the study you are talking about, the exposure is continuing, so that it's a more complicated situation than if dust is going in and it's coming out simultaneously. If you take an exposed group in a factory who then leave at various stages, you've just got the elimination problem. But, I mean it's a difficult problem however you do it.

Q. But overall you do agree that there is an elimination process? You are having problems measuring it, but there is an elimination process?

30 A. Yes, and that has certainly been demonstrated in animals.

Q. While we're on this same paper, and again at

5 Q. (cont'd.) page 187, just by way of clarification, at the end of that paragraph on measures of exposure and the weighting of exposure, I read a sentence here, and I quote, "Thus exposure twenty-five years ago is considered five times as important as exposure five years ago".

A. Yes.

10 Q. Is it possible for me to take your number of ten point eight of 1951, referred to by Mr. Hardy just now, readjust it to thirty-two point four because of the new measurement techniques as demonstrated by Dr. Peto, and what happens if I apply this approach to those numbers?

15 A. Well, what happens is that if we can, if we turn back to tab eight, for example, in table eight on page 107, then except for the adjustment due to the counting method, that is of the ten point eight to thirty-two point four...

Q. Yes.

20 A. ...except for that, we've already done what you have suggested, because we've got models fitted with no elimination, and that is giving the 1951 exposure the weighting because it was thirty years previously. So the only thing you have to do to the figures in table eight, in the righthand half of table eight where we've got fiber counts, is to multiply them by two or three to make them correspond to graticule counting.

25 Q. The numbers in the left column of table eight?

A. No, the ones in the righthand column.

Q. And multiply by whatever difference there is between the old numbers and the adjusted numbers, given the new measurement possibilities?

A. Yes, correct.

30 Q. Always in tab eight, the question of clarification again, on page 100, "fiber counts were not

Q. (cont'd.) "available for 1951 to 1960, but thermal precipitator particle counts, etc., were available".

It's on the righthand side.

Could you describe the thermal precipitator method?

A. I think it would probably be best if you got a dust sampling expert to do that. I would probably confuse things more than clarify them.

Q. In that same paragraph, I would also like to know from you why the counts for 1956 to 1960 were taken to be the same as 1961 to 1965?

A. That is because there was known to have been no major changes in the layout of the factory, no new ventilation equipment had been introduced. In other words, that period was considered to be a period when nothing very much happened to change dust levels.

Q. This was investigated?

A. What do you mean by investigated?

Q. This was assessed on the premises, this assumption?

A. Oh, yes. Yes. I mean this whole work was done in conjunction with people who had been at this factory throughout.

Q. You stated in answer to a question from Mr. Laskin that it was extremely important to have knowledge of dose in the examination of records. Can you elaborate on that statement?

A. Yes. I can't recall exactly which statement you refer to. You mean examination of records, did you say? The way I expressed it...I guess we were probably talking about dose-response relationships. Yes.

No, I'm sorry, I can't really recall Mr. Laskin's question, but maybe we were talking about a different aspect,

A. (cont'd.) but obviously if we are talking about dose-response relationship...

Q. It was in that context.

A. It wasn't?

Q. It was, yes.

A. Ah, yes. Right. Well, I mean, the point I made in my opening part was that given that we know asbestos is a dangerous material, then the next step is to construct dose-response relationships in order to use those to guide future or present policy. So we need dose-response relationships, and obviously if we need dose-response relationships, then we need measures of dose that are as accurate as possible.

Q. These would be necessary to serve as a guide to determine whatever hygiene standards are to be determined, for policy decisions?

A. Yes.

Q. In that respect, how would you qualify Dr. Selikoff's work as a tool to determine policy?

A. Well, the main thing that Dr. Selikoff has shown is a number of work situations where there has been increased mortality, often very much increased. He has shown the smoking/asbestos interaction that we've been talking about earlier, but he has given very little data on actual dose in fiber terms. Therefore, it's difficult to use it in any quantitative sense for deciding on what dust levels might be proven to aim for.

Q. Finally, referring to tab fifteen, the friction materials plant work, would you agree that there is now a thirty year vision of the situation in that plant at dust levels which compare to dust levels which are common in the industry, at least in Quebec and in Ontario, for the past...so you do have a thirty year period where these levels are comparable?

A. Well, we have a thirty year period to...

Q. I think fifty-one...

5 A. ..to levels which are in, historical terms, quite moderate. I mean I've got knowledge of what levels are in Ontario, so I can't say whether they are comparable to that or not, but it's thirty years exposure to what are, in historical terms, quite low levels, moderate levels, and are certainly a lot nearer to the sort of standards that are spoken about today than most asbestos factories were operating at thirty years ago.

10 Q. Given the results and your conclusions in this work, wouldn't these be in the area of hygiene standards which would be acceptable?

15 A. Well, there's two qualifications to make to that, and one is that...and it isn't given in this paper because this is work we've done in the last few months...but I did think I mentioned it yesterday, I'm not sure. I think maybe it was in some of those slides at the end that I skipped over, but we then...since then we have worked out cumulative doses for lung cancer cases and control men employed at the same time in the factory. The cumulative doses are very, very low. For example, it is about...if I can find the paper...you haven't got this, but if I can just look at my own notes...yes, about half the lung cancer cases I've got a cumulative exposure of less than ten fiber years per ML, which is a very low level. This is because a lot of these workers, although there is a thirty year latency, a lot of them didn't stay at the factory for very long. There are only four who reached...four out of a hundred lung cancers who got up to a hundred fiber years.

25 I think looking at table one on tab fifteen, on page ten dot six dot two, the fact that only four reached a hundred fiber years will be surprising. It certainly was a surprise to us when we did this, and the consequence of this is that the dose-response relationship isn't estimated very

30

A. (cont'd.) precisely.

5 If I can go to the blackboard...this is dose and this is lung cancer rate in comparison with it...lung cancer SMR in comparison with a hundred in the general population, and then a dose-response relationship looks like that.

10 McDonald used a graph like that in some of his papers and if you look at the Lyon paper, which we were talking about earlier, where he has done some fiber count estimations, then we are talking here about, say, an SMR of five hundred.

McDonald has graphs that go to five hundred for SMR, with points here, and the dose here in fiber years is compared with what I'm talking about, which is some huge figure something like maybe nine thousand fiber years.

15 If you recall the figure I gave yesterday on the slide where I spoke of a hundred and four at a hundred, that is a sort of extrapolation from this huge range of data.

Now because we've got this huge range, point seven here, he demonstrated that a dose-response relationship existed.

20 Now, data I'm talking about all occurs down here. I think it is reasonably obvious that if you are trying to estimate where to put the line through some data, what you really want are data points way over here. If you've only got a little bit of data down here, you can't fit a dose-response relationship very accurately. And that's the situation we're in at this factory, and I won't say unfortunately because it was very fortunate for the people who worked there, of course, but it meant that the upper limit of this slope was a hundred... corresponded to a hundred and eighty at a hundred here, so the upper limit of our data was like this, and the actual dust estimate would be very similar to the McDonald line.

30 If you contrast this to McDonald, looking at his upper limit, it's something like this - it looks quite close to

A. (cont'd.) the line.

So, it's rather a long answer to your question, I'm afraid, but the two points are certainly in this study. There was no evidence of an excess lung cancer risk, and that is the point that you were getting at, I think, but there is also insufficient evidence to contradict...I'm sorry...there is insufficient evidence to contradict a non-negligible increase in lung cancer.

Q. You mentioned yesterday, and it's the final point, that at one twenty-five it becomes perhaps detectable in incidence, is that correct?

A. Oh, that was just a figure I threw out for illustration. I mean, what is detectable depends on how big a group you've got, but I think I was talking about, mentioned one twenty-five you would have to have at least an expectation of a hundred lung cancer deaths, and I think I mentioned that most studies that have been done historically have worked with groups where there maybe haven't been as many of that. I mean, some of them have had more. But it does correspond to quite a big group of men, followed-up for quite a long period of time, and I was making the point that it is going to be difficult to detect that type of increase.

Q. Dr. Well, when testifying here on the question of threshold TLV's, mentioned the principle of acceptability of risk on the one hand, and also the problem of detection on the other hand, as two of the items that had to be taken into account. He mentioned the figure at one-fifty for detection.

Do you have any comment on that?

A. You mean he was saying that unless the rate was as high as one-fifty it would be difficult to detect?

Q. Yes.

A. Yes, well, that's, you know, a similar point

5 A. (cont'd.) to the one I was making. One can't actually, you know, state any definite figure because it depends on the size of the population, but I agree with him that one... even an increase up to one-fifty would be quite difficult to detect. It would need a big group followed-up for a long time.

MR. BAZIN: Thank you.

DR. DUPRE: Dr. Uffen, any questions?

DR. UFFEN: No, I had my opportunity yesterday.

DR. DUPRE: Dr. Mustard?

10 DR. MUSTARD: Yes, Mr. Chairman.

I would like to go to and ask you some questions related to data which is in a number of the tabs.

15 Related to cancer in other sites, particularly gastrointestinal cancer, your table one in tab six indicates for gastrointestinal cancer, particularly in those given severe exposure, that the observed is greater than the expected, but the figures in brackets for mesothelioma, if you subtract them out, decrease those figures?

MR. BERRY: Yes.

20 DR. MUSTARD: I would presume that what's left is probably not statistically significant, but the question is, how do you control in the expected for mesothelioma? Have you controlled for that in terms of gastrointestinal cancer?

25 My reason for asking you a question about the table is, that presumably you pulled the...somewhere in the classification they were given as gastrointestinal cancers, but your subsequent digging found out that they were mesotheliomas, is that correct?

MR. BERRY: Yes.

30 DR. MUSTARD: In the case of the expected, you are dealing with the system out there making a diagnosis of gastrointestinal cancer, is there a possibility in that expected figure that you have got some mesotheliomas hidden in it, in

DR. MUSTARD: (contd.) terms of where you are calculating from national statistics?

5 MR. BERRY: Yes, that is correct. In fact we know, in fact, that there are some mesotheliomas hidden in it because the expected is for the whole country, and therefore does actually include these people...a very small percentage of it, of course.

10 And we would also expect that there would be mesotheliomas hidden in the expected figures, based on other asbestos-exposed situations. But we do...there is a fairly good idea of how many mesotheliomas are occurring in the U.K., and it's around about three or four hundred per annum, I think, at the moment, and of course a lot of those are pleural anyway, so that the number that are hidden is quite small or very small in relation to the total number given that diagnosis.

15 DR. MUSTARD: So if I look at that table can I draw any conclusion as to whether forms of gastrointestinal cancer other than mesothelioma have been influenced by the asbestos exposure?

20 MR. BERRY: Well, the conclusion you can draw is that it would be fair to compare, if we look at severe- less than two years, the twenty minus the six, fourteen, with a nine point five, and eleven with eight point two, so we've got there twenty-five compared with seventeen point seven, which I wouldn't, as you observed, I doubt if that would be significant. But it is raised and there have been..we've
25 also got other cancers which are raised as well, cancers other than the GI tract.

30 I think actually from this study alone, one couldn't make any significant statement on whether GI cancers had been increased, but there have been other studies and the overall effect of them is that there is an increase in gastrointestinal cancer and other cancers, and I heard a

5 MR. BERRY: (cont'd.) hypothesis put forward last week at a meeting I was at, by a Professor Goldsworthy, I think his name was, from Israel, who looked at a number of these studies and the gist of what he was saying was that the raised incidence of GI cancers was no higher than that of other cancers, but because GI cancer is quite a common cause of death, these have been more noticeable.

10 But looking at all studies together, then there is evidence of an increase in both GI and other cancers, because not everybody has done as much digging as Dr. Newhouse.

15 DR. MUSTARD: Can I pursue this a bit further? If you turn to table six, for the females, and look at the comparisons with the mesotheliomas, you are dealing with an observed over expected, in the severe exposures...I take it you combined the two groups together...?

MR. BERRY: Yes.

DR. MUSTARD: ...of a magnitude of two to three times the observed over the expected, for the females?

20 MR. BERRY: Yes, that's right. Yes. You've got seventeen compared with eight point three. Yes, I would think that would be significant.

DR. MUSTARD: Is one to run the risk of inferring from this that females might be more susceptible than males, or that their habits in the workplace are different from the males? Or is that just a chance observation?

25 MR. BERRY: Well, we have discussed earlier the lung cancer is raised in females relative to the expectation, and maybe this is the same sort of thing we discussed earlier. It could be that they had higher exposures, but no evidence on that point.

30 MR. MUSTARD: This raises, the reason I bring this up is that when we heard from Dr. Weill last week, his classification of digestive cancers, his asbestos-exposed workers

5 DR. MUSTARD: (cont'd.) had an observed over expected which was less than a hundred. I believe in some of the groups it was quite low, standardized mortality ratio. I see the opposite here, which I find very difficult to cope with.

10 One of the things I detect in looking at this, there may be some male-female differences which obviously, you have indicated, may hold for other cancers as well, but this raises a very basic question. How do you determine the expected? You've got a group of workers who have been exposed to something. What's the basis by which all of you people working as biostatisticians determine the expected?

I raise this particularly because in your country you have had a tendency to classify deaths by social class.

15 MR. BERRY: Yes.

20 DR. MUSTARD: The mortality for some disease, including some of the ones we are looking at here, are much higher in the social class of four and five, which are the unskilled and semi-skilled. Do you make any kind of adjustment, therefore, in the expected, for the characteristics of your work force - race, sex, socioeconomic class?

Perhaps you could explain it for me.

25 MR. BERRY: Well, we certainly take account of sex, but all these expected figures are based on the death rates for England and Wales, taking account of age and taking account of the period of time that these people were alive, because the death rates have changed over time.

30 As you say, there are differences due to social class, and there are also differences between urban and rural workers. We haven't taken it into account and the approach that I adopt over this is that because of uncertainties as to whether the population for England and Wales, as a whole, is appropriate for a working group, and there is one big difference which is sometimes called the healthy worker effect, one of the

5 MR. BERRY: (cont'd.) features of which is that those people, that the national population includes unemployed people, some of whom are unemployed because of their health. It includes chronically sick people, for example, who wouldn't be able to keep a job, and therefore, in a group of people who are working in a factory doing quite demanding work, you would expect them to be fitter than the general population, and this has indeed been demonstrated, particularly over a short period of time.

10 For example, if you take a group working in a factory and look at them over the next ten years from when they started, you could find that their overall standardized mortality ratio was less than...well, was about, say, sixty or seventy percent. So we've got this problem as well as the one that you have mentioned, which are difficult to take exact account of, 15 so the approach that I have followed is to rely on doing comparisons within a study and look at dose-response in some sense, within a study. So we are looking...if in table six, which we were looking at...in judging whether an effect can be attributed to the exposure, we have to look and see whether it 20 shows up in the severely-exposed group and not in the low-to-moderate group.

DR. MUSTARD: That gives you some confidence that there is probably an effect?

MR. BERRY: Yes.

25 DR. MUSTARD: But it doesn't give you much of an index about the true nature of the influence of the factor on the population, because you've got the background noise when you get down to the low exposure levels, etc., is that right?

30 MR. BERRY: Well, it still gives you a measure of the effect of the occupation if, say, one contrasted a severe group with a low-to-moderate group where the effect is going to be much less. It does mean that we can't, perhaps,

5 MR. BERRY: (cont'd.) conclude that the low-to-moderate group, because they show no increase of observed with expected...actually in the case we are looking at, they do...but if we had the case where they didn't, one couldn't necessarily conclude that there was nothing in the working environment which was causing mortality.

10 When we compare the severe with the low-to-moderate, we can then show that there is something which is certainly increasing the mortality. And that's one reason why, if we go back to the study of the friction materials, which is tab sixteen, I think, why it was important to do more work on the data than we did in tab sixteen, where we just compared observed with expected. Because there the only dose-response relationship we had was with length of time worked. There was no account of job done, and so it could have been that the observed equal 15 the expected because there was an effect of the exposure which cancelled out some other effect which was reducing the death rate.

20 Well, now we have done further work which, as we are discussing, and doesn't lead to a dose-response relationship.

25 DR. MUSTARD: Can I assume from your answer in terms of how you calculated the expected, that based on the evidence you have that your expected is probably an overestimate from the group that you are comparing against? I'm concerned about which weighting one gives to the expected...

MR. BERRY: Right.

30 DR. MUSTARD: You've said it's for the total population, you have the healthy-worker effect versus the full mix of the population.

MR. BERRY: Yes. I think with asbestos exposure, because of the latency, a lot of the health-worker effect has been removed as the main effect. The main way the healthy-worker effect shows up is in the mortality of people who have just

MR. BERRY: (contd.) got a job.

5 If one follows these people up over a period of time, the SMR's approach a hundred from below, and there was a paper by Fox and Cordue, which showed this very well, and the healthy-worker effect had largely disappeared by about fifteen years after the worker was employed.

10 Fortuitously, the analysis of asbestos workers don't need to consider the initial period, because we know there is a latency and in the work I report we are always looking at mortality more than ten years after first exposure. So a lot of the healthy-worker effect has been eliminated in any case.

15 Because we've still got, you see, if somebody gets a job and then becomes disabled for some reason, and leaves, is not able to hold a job down, we've still got them in our mortality study.

20 But no, it wouldn't be complete because there would be a certain percentage of people who were never capable of holding down jobs..they've got certain congenital malformations and people with Down's Syndrome, things like that...but that is a quite small minority, I think.

25 DR. MUSTARD: I would like you to go to the board and just tell us how you do the calculations for the observed versus the expected, and particularly take an example that you are doing a cohort followup in the workplace and let us say that you can only followup three-quarters of the cases that have been exposed to asbestos, in terms of mortality.

Do you understand my question?

MR. BERRY: You meant twenty-five percent non-traced?

DR. MUSTARD: Yes. Can you show how that influences the observed versus expected calculations?

30 MR. BERRY: Yes.

What I'm drawing here is a grid which has got

5 MR. BERRY: (cont'd.) one axis time, the calendar year, and on the other, age. I'm drawing this grid in five year increments, and I'm doing this because the mortality rate, the death rates, as published by the Registrar General for England and Wales, give death rates by five year age groups, and they have also been produced for five year periods of time. That is to say, we know what the death rate is of people aged thirty to thirty-four in the period 1950 to 1954...actually it would be 1951 to 1955, and we know what it was ten years later.

10 Now what we do is, we take...let us take a man who joined an asbestos factory when he is aged twenty-eight, and it happens to be 1948. Now, he is then in our study. Whatever he does afterwards, he is in the study, whether he leaves the factory or what have you, he is in the study.

15 As time goes by, we obviously move along this axis, and the man also gets older, so he goes across this diagram in that way.

20 Now he leaves the study at some point, and there are three reasons why he might leave. One is that he dies at that date, another is that...over here in 1980, that that is the time that we've done the followup to, so he hasn't actually left the study, but we've only got observations up to that point. So we've got deceased people and alive people, and then we've got the other group who we try and make as few as possible, of people who we lose track of.

25 What we do for those is, we keep them in this study up to the date that we lose track of them. So we might know, for example, that the person was at the factory. We might have from factory records that he was there for twenty years, so we know that he was alive for that twenty years, but then he left the factory and for some reason we can't trace him. Then we take him out of the study at that point and say, effectively say, we don't know anything about what

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MR. BERRY: (cont'd.) happened to him thereafter.

Now we draw lines like this for everybody in the study...when I say, we don't actually draw them, of course...but this is the nature of the calculation, and for each of these squares we measure the lengths of these lines, which is the number of years that the man was aged thirty to thirty-four, in the period 1951 to 1955, and we add this up over all the people. For that reason it's sometimes...this method is often called the man-years approach, and we end up with a number of man years for each of these squares, which we then multiply by the death rate which we've got from the Registrar General's figures. We multiply years at risk by death rates, and that gives us the expected number of deaths.

So the one point you made was that people who had left...we only filled them into this calculation for the period of time that we know about them, and of course if we lose track of somebody this is a possible source of bias, because it may be that we lose track of people more readily if they die, say, or maybe if they are living, but one common reason for losing track of people is because they emigrate. When I say common, I think it's in the friction materials it was around about...I've forgotten, but maybe it was two or three percent.

Of course, we know, or at least strongly suspected that people who emigrated are fitter people, because often where they are going to, you have to have a medical examination anyway to get a visa.

So that losing track of people has a possibility of introducing a bias, so we try and make that as low as possible. If we had lost track of twenty-five percent of people, there would be obviously more room for bias than if one lost track of one percent.

DR. MUSTARD: Which way could that bias go?

5 MR. BERRY: Well, if we are talking about emigrations, saying that the people who left are fitter, and therefore we are cutting out people who have a lower death rate, so that we are increasing the mortality, because we have taken people out who are more likely to have survived.

DR. MUSTARD: Increasing the mortality on the observed side?

MR. BERRY: Yes, that's right.

10 DR. MUSTARD: But supposing you are within a country in which there wasn't much emigration, but you just simply couldn't track them down, if you had that factor, you didn't know what the factor was that was...

15 MR. BERRY: Right. Well, that could go either way, and it might be that one lost track of them because they had died, say. Maybe in some situations you lose track of them because they leave the factory, they've left the factory because they are disabled, and they are therefore more likely to die.

20 Often, of course, one can't track them down because the identification details in the file of the factory are insufficient, and that's...the studies we have done in the United Kingdom have that characteristic. If there's good identification details in the factory, then we will achieve at least a ninety-nine percent followup, as we did in this friction plant data.

25 DR. MUSTARD: All right. Then come the conclusions that in our giving weight to the various studies that we have to hear, that we should give the greatest weight, everything else being equal, to those studies that have the best followup?

MR. BERRY: Yes.

30 DR. MUSTARD: The weaker the followup, the greater the risk of bias?

MR. BERRY: Yes.

DR. MUSTARD: In either direction?

MR. BERRY: Yes.

5 DR. MUSTARD: Okay. That helps straighten out that point.

I would like to turn to tab thirteen, if I may, which I believe is the asbestos dust in the gas-mask factory document, and turn to page 651 and see if I understand figure four.

10 MR. BERRY: Oh, well I don't. That's a terrible figure, but the other authors like it.

DR. MUSTARD: The thing going along the horizontal axis is simply just, each case has it's own line, is that what I understand? It's...?

MR. BERRY: Well...

15 DR. MUSTARD: It says, individual cases of mesothelioma.

MR. BERRY: Yes, and that's one of the things I don't like about this figure.

DR. MUSTARD: I don't understand it.

20 MR. BERRY: It's just a way of spreading things out a little, that's all. The horizontal axis really has no meaning other than to spread things out...well, it has meaning in dividing the Nottingham cases from the others, but...

DR. MUSTARD: I see that.

25 MR. BERRY: ...otherwise it's got no meaning.

DR. MUSTARD: Now, then, as I look at that, the vertical axis does have meaning. That's clear.

MR. BERRY: Yes.

30 DR. MUSTARD: When I go through that, I see mesotheliomas...the black squares...it could be my glasses... are right down at the same level of fiber count as the CVA controls, and I presume this means the nonmesotheliomas are also controls, is that right? Or just the CVA's are controls?

MR. BERRY: Just the CVA's, I think, it would be best to take them as.

5 DR. MUSTARD: I find that very intriguing. I realize that there are all kinds of problems with it, but it's raised a question being asked earlier about the background exposure to asbestos fibers and the problems which develop, and just looking at that table one would have to assume, on the basis of the data, that there can be people who die without evidence of mesothelioma, who have in their lungs at the time of death the same number of fibers as people who die from mesotheliomas?

15 MR. BERRY: Yes. Yes, that is correct. One figure we were looking at earlier, which I prefer to illustrate this point, is from tab nine, figure two or even figure three, which shows the same sort of thing. We've got some control people who have got...well, two points. We've got some mesotheliomas deaths with relatively low levels, and we've got control people with relatively high levels, and there is an overlap even though there is quite a difference.

20 If you look on page 194 of tab nine, figure three, there's a difference between the control material in the mesotheliomas of about tenfold, I think it is...that is on our bridge, the mesotheliomas have got ten times as much amphibole in their lungs as controls. But there is quite a lot of overlap. They are not two quite distinct groups.

25 DR. MUSTARD: In animal experiments where you have standardized the dose and track the animals, do you get this same kind of experience that exposing a hundred animals to a standard dose of fibers, only X percent develop pathology?

MR. BERRY: Yes.

30 DR. MUSTARD: Do you have any explanation for why that difference occurs?

MR. BERRY: You mean of why some...

DR. MUSTARD: Are more susceptible than others?

5 MR. BERRY: No, not really. I mean, one thing is biological variation, which of course as a statistician I am very fond of. If it didn't exist, I would have to change my profession.

10 And that is to say that within a biological population, different individuals are differing in quite a lot of ways, and I mean we can leave exposure out of things and just say if we kept a colony of rats and didn't expose them to anything, looked after them all very well, gave them all exactly the same food, etc., some would die young and some would die old. No doubt if we knew a lot more than we do now, one could measure a lot of factors on a rat which might enable one to predict how long they were going to live, but I don't think we are anywhere near that situation, and I think it's the same when you expose a homogeneous group of rats to a dust, that some will develop a tumor quickly and some will take a long time, and some will die of something else without having developed a tumor.

15 DR. MUSTARD: I take it then that one could be suspicious in looking at this data that the human data that's coming in, I realize there are a lot of other variables in this fiber count business, could imply that there is some evidence of variation among human beings in terms of the susceptibility, given they were all within, saying within an experiment they were given a standard dose of asbestos fibers?

20 MR. BERRY: Yes, there must be differences of susceptibility, yes.

DR. MUSTARD: Okay...

25 MR. BERRY: I suppose this comes out very clearly with smoking data.

30 DR. MUSTARD: If I go back to tab six, page 55,

5 DR. MUSTARD: (cont'd.) in the first paragraph,
in the third sentence, there is a statement: "Asbestos-related
disease is rarely, if ever, manifest in those
dying within ten years of first exposure".
Could I ask you what is the basis for this
statement, the evidence?

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15 THE FOREGOING WAS PREPARED
FROM THE TAPED RECORDINGS
OF THE INQUIRY PROCEEDINGS

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5 MR. BERRY: Yes; it's based on looking at the mortality in that person ten years after first exposure, and observing that there are no mesotheliomas, or, if there are, it's just an isolated one.

10 In fact, the only mesotheliomas we've had in this study, in less than ten years, occurred in a lagger; and a lot of the laggings, we know, have had prior exposure elsewhere. So it's based on observing that the mesotheliomas never occur in less than ten years.

15 It's based on observing that the incidence of lung cancer deaths in the first ten years is -- that there's no excess, even in groups which, soon after that, or certainly after fifteen years, show quite a marked increase.

20 DR. MUSTARD: But it's not based on a post-mortem study, therefore, of individuals dying during -- any time after their exposure to asbestosis and detailed analysis of the lungs to see if there's any fibrosis, which would be asbestosis; it's based on death related to mesothelioma. It's not really based on a strict, specific diagnosis of asbestosis at a tissue level?

MR. BERRY: That's correct; yes.

25 DR. DUPRE: Before you let that one go, Dr. Mustard, I heard your response to Miss Jolie's question, which, if I remember right, was that, for compensation purposes, a criterion that required ten years of occupational exposure would be, in your view, a very strict criterion.

Now, how does that response that you gave to Miss Jolie square with the observation about which Dr. Mustard has been asking?

30 MR. BERRY: The observation Dr. Mustard's been raising is the -- how soon the death occurs; whereas, if I understood Miss Jolie correctly, she was talking about the

5 MR. BERRY: (cont'd.) total length of exposure. If she'd been talking about deaths due to lung cancer which occurred within the first ten years after first exposure, if they were excluded, then I would not think that was -- I would think that was quite reasonable. But I think she was talking about people who'd had less than ten years' exposure, but then developed a lung cancer, say, twenty years later.

10 And it was particularly for mesotheliomas, where I regard that as a very stringent test.

DR. MUSTARD: Can I have one final question.

15 From the documents you've presented and your answers to questions today and yesterday, I would take it that, in the United Kingdom, asbestos was recognized as a health hazard in terms of public action since around 1932. Is that correct?

MR. BERRY: Yes. There was a famous report in 1931, it was, which led to a set of regulations, referred to as the asbestos regulations of 1931; yes.

20 DR. MUSTARD: And from that time on, then, it has been systematically studied within that country?

MR. BERRY: Not really, no, because, soon after that, there was the war, of course, and there was no studies going on in that time; and also, I think it tended to be assumed that, because of the 1931 regulations, that the problem had been solved.

25 The 1931 regulations were mainly concerned with asbestosis; though I think there were indications at that time that lung cancer was a problem, but it wasn't really accepted, I don't think, at that time, and it wasn't **really** until about 19... -- the 1950's, and I'm thinking particularly of a paper by Dahl in 1955, which was the first paper on the Rochdale factory, which we've spent so much time talking about, when the raised

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MR. BERRY: (cont'd) incidence of lung cancer mortality was conclusively brought out.

And that paper led to a lot of work starting and to research being done, and another important paper was by -- which I referred to yesterday -- was by Wagner, Sleggs, and Marchand, in South Africa, on showing the relationship between asbestos and mesothelioma. Again, that paper led to a lot of work.

So, certainly, it wasn't systematically studied following the 1931 regulations; there was a period of about twenty years when there was very little research going on.

DR. MUSTARD: And the next set of stimulus came from the identification of the lung cancer story?

MR. BERRY: Yes.

DR. MUSTARD: When did that lead to the next major shift in public policy for the control?

MR. BERRY: Well, as far as actually any legislation was concerned, not until 1969, although there were changes within factories. Again, some of this, I showed you yesterday.

The factories, because of that work, were improving their hygiene, but not -- there was no legislation actually making them do this at that time, different from the 1931 regulations.

DR. DUPRE: Mr. Berry, I'd like to address your paper that is on tab number 4; that is the hygiene standards paper.

Now, on page 147, in the righthand column, under "Discussion" -- and bearing in mind that you wrote this paper -- or, at least, the paper was published in 1973, which is eight years ago -- but in the point under discussion, at the bottom of the page, you give some -- I take it, it's professional opinions as to the kind of data that were available then, and you point,

DR. DUPRE: (cont'd) among other things:

"There is a serious dearth of data on the relationship between ill health and exposure to different levels of different types of asbestos." You continue, over on page 148:

"Data are needed representing different conditions in different countries. Data are also needed on the relationships among the different stages and types of asbestos-produced disease. Some evidence is available which indicates that the excess risk of lung cancer occurs mainly in those with asbestosis ... but the position in relation to pleural and peritoneal mesotheliomas is uncertain," et cetera.

My question is really this: if you were writing this paper in 1981, to what extent would you modify the observations that you have made in the 1973 paper about the relative paucity of data, and about the extent to which data needs that you mentioned in '73, perhaps, are now being filled by 1981?

MR. BERRY: Well, certainly since 1973, there's been a lot more data looking at the problem from a dose-response point of view, and a lot of these have been from the cancer viewpoint, which is, I think, more important than the asbestosis.

When this paper was written, I don't think there was any dose-response data on relating lung cancer to quantitative dust measurements. So, certainly, one would have to modify that statement.

One would still not be able to say the situation is very satisfactory; one would have to point out the limitations of the dose-response, particularly the point which we've spent quite a bit of time on: the changes ways of measuring

MR. BERRY: (cont'd) dose, of measuring dust concentrations.

5 But, certainly, there has been quite a lot produced since 1973.

10 DR. DUPRE: And with respect, in particular, to the second sentence in the righthand column on page 148, the one that begins with "Some evidence is available ..." et cetera, is there anything, with the passage of eight years, that would lead you to add or modify, in terms of the view you state there about what is available, both with respect to the risk of lung cancer in relation to asbestosis and in relation to pleural and peritoneal mesotheliomas?

15 MR. BERRY: Yes. I think I would write that sentence differently; in fact, it probably isn't phrased correctly for 1973, because the Knox paper which is referred to, that actually showed that, if one took the difference between the observed and **expected** lung cancers, the figure you got was very nearly equal to the number who had some signs of asbestosis, which isn't quite the same as saying that the excess risk was in those who had that disease.

20 And there has -- there have been one or two papers published since then which have shown an increased lung cancer risk in the absence of asbestosis, although some people might query what is meant by an absence of asbestosis. And there hasn't been a great deal of work to address on this issue in
25 either direction.

30 I think my main opinion at the moment is that asbestosis and lung cancer both occur due to exposure; those who develop asbestosis, on average, will have had higher exposures than those who don't; and that would be true of lung cancer also. So that you will tend to observe the two diseases going

5 MR. BERRY: (cont'd) together, but that is not to say there is any causative mechanism; that is, that the actual fibrosis, the fibrotic reaction, actually then causes the lung cancer. I don't think there's any evidence to show that that's the case.

10 And you asked about mesotheliomas. I haven't really regarded this as terribly important in mesotheliomas, though maybe I've been wrong not to take that attitude; but, because mesotheliomas have been rare, it has seemed justifiable to assume that they are a result of the asbestos exposure, if one knows that a man has been exposed, and it's, therefore, irrelevant to consider whether he's got asbestosis or not.

15 I mean, this point is, I think, mainly considered of importance for compensation purposes. And one can recognize that there's a difficulty in transferring the results of epidemiology, which are all epidemiologies concerned with group -- the situation within groups -- it's difficult transferring that to a compensation situation where you're having to make an individual decision on a particular man.

20 And we know, of course, that lung cancer is quite common. I don't think it's as common in Canada as in the U.K., but I still expect it's quite a common cause of death. And, therefore, in an individual worker, one can't say whether his lung cancer is due to his exposure or would have occurred in any case.

25 The only thing I think one can say is that, in a particular group, because they've been studied epidemiologically, that may/^{be}seventy-five per cent are asbestos-related; that is, if we've got an SMR of four hundred in a group, then we might be able to say that seventy-five per cent are asbestos-related.

30 In a situation where the doses are a lot less, which is the case in the future, one hopes, then the position

5 MR. BERRY: (cont'd) would be even more difficult, because then you would be in the position that lung cancers would still be occurring; some of these would no doubt be due to exposure, but it might be that, say, only, let us say, one-third instead of three-quarters -- only one-third were due to the exposure.

10 And I -- you know, within a compensation situation, I don't really know how to resolve this. And I can see that, from that viewpoint, using asbestosis as a guide, has quite a lot to say for it, because it indicates that those people at least had sufficient exposure for some reaction to take place, which people, like the pathologists, prefer to say is due to the asbestos.

15 And, of course, one's in an even worse position with other types of cancer.

20 DR. DUPRE: Could I ask you the following. Dr. Weill, when he was with us, made the following observation about standards, being, of course, very careful to say that he does not have any specific numerical standards in mind; but the point that he made was that a standard that might be low enough to prevent excess mortality from lung cancer would not be a standard that is low enough to preclude asbestosis.

25 Or, to put it another way around, a standard that precludes asbestosis will also automatically preclude lung cancer.

Do you have any view on that opinion?

30 MR. BERRY: Yes. I think, on the whole, I would agree with that, because asbestosis -- particularly if we're talking about diagnosing it, either in life or at post mortem -- it is detected in quite a high proportion of the work force, more than developed lung cancer, so that I think it is a reasonable supposition to think that, if one reduced dust levels so

5 MR. BERRY: (cont'd) that asbestosis didn't occur at all, then one would certainly have mainly controlled lung cancer, but I wouldn't say one had controlled it completely.

There are one or two studies which indicate otherwise, but there's very little.

10 DR. DUPRE: Now, can we just extend that. What would you think of the following: that a standard that is low enough to preclude both asbestosis and excess mortality from lung cancer is a standard that is still not low enough to preclude mesothelioma?

15 MR. BERRY: Yes. Well, of course, I don't -- maybe I'm slightly modifying my answer to the last question, but I don't accept that one can completely preclude lung cancer, but only that it will get to not very much above expectation.

20 And I would say the same for mesothelioma, except, there, we don't have much background to confuse the issue; and, therefore, we can recognize mesothelioma -- we can recognize, when a mesothelioma occurs, that it is due to the exposure, even though that is low exposure. And it's probably a valid assumption to say it is due to that low exposure. You can't do that with lung cancer.

25 And because of the detection of an excess, we were talking about earlier, it means that we might have a standard where we certainly can't detect lung cancer is raised, but where we do see mesotheliomas. But it might be that we're getting a similar number of excess lung cancers, but we just can't measure them.

30 DR. DUPRE: Again, on that page 148, you talk, of course, simply about the importance of data for the sake of international comparison, and I reflect, once again, on tab 9, page 193, which has caused a fair amount of questioning around

DR. DUPRE: (cont'd.) here.

Are there any studies in other countries that give you any kind of comparison as to what you would find in a non-asbestos population in terms of asbestos particles in the lungs?

MR. BERRY: Not in reasonable size populations, that I'm aware of, no. There are two other people, I can think of, in the world, who are -- would be as capable of doing this as Dr. Pooley is, and one of them's in the United States and the other's in France, and they've got similar equipment, but they don't do this type of study on -- I won't say a large collection, because we're only talking of a hundred and fifty or so people in this study, although the study we were doing in 1977 has got bigger numbers, but I don't think it's even been done on moderately sized groups.

DR. DUPRE: Counsel, any final questions?

MR. LASKIN: I don't think so, thanks, Mr. Chairman. I think we've kept Mr. Berry on the witness stand long enough.

DR. DUPRE: Well, Mr. Berry, we are all most grateful, indeed. You came a long distance, at considerable inconvenience to yourself, and I must say that you have certainly won every accolade I could think of as a marvellous teacher. Thank you, indeed.

MR. BERRY: Thank you, Mr. Chairman.

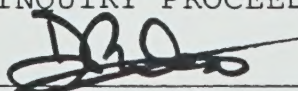
MR. LASKIN: I think, Mr. Chairman, we're adjourned until next Wednesday ---

DR. DUPRE: We are adjourned until, I presume -- unless Miss Kahn corrects me to the contrary -- we are now adjourned until 10:00 a.m., on Wednesday, June 24th; is that correct? At which time, Dr. Corbett McDonald will be here.

INQUIRY ADJOURNED

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THE FOREGOING WAS PREPARED
FROM THE TAPED RECORDINGS
OF THE INQUIRY PROCEEDINGS


DEREK WEST

